STUDY OF CLINICAL AND ENDOSCOPIC PROFILE OF UPPER GASTROINTESTINAL BLEED IN A TERTIARY LEVEL HOSPITAL – GRH, MADURAI

Dissertation submitted for

MD DEGREE (BRANCH 1) GENERAL MEDICINE

APRIL 2017

THE TAMILNADU DR.M.G.R

MEDICAL UNIVERSITY

CHENNAI – tamil nadu

CERTIFICATE FROM THE DEAN

This is to certify that this dissertation entitled" **STUDY OF CLINICAL AND ENDOSCOPIC PROFILE OF UPPER GASTROINTESTINAL BLEED IN A TERTIARY LEVEL HOSPITAL – GRH, MADURAI** is the bonafide work of **Dr.PUGAZHVANAN.C.R** in partial fulfillment of the university regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai, for M.D General Medicine Branch I examination to be held in **April 2017.**

Dr. VAIRAMUTHU RAJU MD.

THE DEAN,

Madurai Medical College,

Madurai.

CERTIFICATE FROM THE HOD

This is to certify that this dissertation entitled "**STUDY OF CLINICAL AND ENDOSCOPIC PROFILE OF UPPER GASTROINTESTINAL BLEED IN A TERTIARY LEVEL HOSPITAL – GRH, MADURAI** is the bonafide work of **Dr.PUGAZHVANAN.C.R** in partial fulfillment of the university regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai, for **M.D General Medicine Branch I** examination to be held in **April 2017**.

DR.V.T.PREM KUMAR,M.D.,

Professor and HOD,

Department Of General Medicine,

Government Rajaji Hospital,

Madurai Medical College,

Madurai.

CERTIFICATE FROM THE GUIDE

This is to certify that this dissertation entitled "**STUDY OF CLINICAL AND ENDOSCOPIC PROFILE OF UPPER GASTROINTESTINAL BLEED IN A TERTIARY LEVEL HOSPITAL – GRH, MADURAI''** is the bonafide work of **Dr. PUGAZHVANAN.C.R** in partial fulfillment of the university regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai, for **M.D General Medicine Branch I** examination to be held in **April 2017.**

DR.R.PRABHAKARAN,M.D.,DLO

Professor of Medicine ,

Department Of General Medicine,

Government Rajaji Hospital,

Madurai Medical College,

Madurai

DECLARATION

I **Dr. PUGAZHVANAN.C.R** declare that, I carried out this work on " **STUDY OF CLINICAL AND ENDOSCOPIC PROFILE OF UPPER GASTROINTESTINAL BLEED IN A TERTIARY LEVEL HOSPITAL – GRH, MADURAI''** at the Department of Medicine, Govt. Rajaji Hospital during the period **January 2016 TO April 2016**. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree or diploma to any other University Board either in India or abroad. This is submitted to The Tamilnadu Dr.M.G.R.Medical University, Chennai in partial fulfillment of the rules and regulations for the M.D degree examination in General Medicine

Place : Madurai

Dr. PUGAZHVANAN.C.R

Date:

ACKNOWLEDGEMENTS

At the outset, I wish to thank our **Dean Dr.M.R.VAIRAMUTHURAJA M.D.,** for permitting me to use the facilities of Madurai Medical College and Government Rajaji Hospital to conduct this study.

My beloved Head of the Department of Medicine **Prof. Dr. V.T.PREMKUMAR M.D.,** has always guided me, by example and valuable words of advice and has encouraged innovative thinking and original research work done by post graduates.

I shall remain eternally grateful to my unit chief **Prof. Dr. R.PRABHAKARAN M.D** who has given me his moral support and encouragement through the conduct of the study and also during my entire postgraduate course.

I also sincerely thank our beloved professors **Dr.R.Balajinathan.MD., Dr.G.Bagialakshmi.M.D., Dr.Natarajan M.D., Dr.J.Sangumani. M.D., and Dr.C.Dharmaraj. M.D.,** for their par excellence clinical teaching and constant support.

I am extremely grateful to our **Prof.Dr. M.KANNAN MD DM;** Head of the department of the Medical gastroenterology , and **DR.R.RAMANI MD DM**; Assistant Professor Department of Medical gastroenterology for their constant support, guidance, cooperation and encouragement to complete this study.

I offer my heartfelt thanks to my unit **Assistant Professors Dr.SYED BAHAVUDEEN HUSSAINI MD, DNB.,** and **Dr.P.SARAVANAN MD**.,for their constant encouragement, timely help and critical suggestions throughout the study and also for making my stay in the unit both informative and pleasurable.

My patients, who form the most integral part of the work, were always kind and Co operative. I pray to God give them courage and strength to endure their illness, hope all of them recover from illness.

I thank my friends and family who have stood by me during my times of need. Their help and support have always been invaluable to me. And last but not the least I would like to thank the Almighty for His grace and blessings without which nothing would have been possible.

TABLE OF CONTENTS

Table of Contents

Introduction

Upper gastrointestinal bleeding (UGIB) is a common gastrointestinal (GI) emergency and mortality rates of 5% to 11% have been reported representing a serious and life-threatening entity, despite advances in diagnosis and treatment.

- The epidemiology of UGIB varies among population and there is a paucity of data on UGIB and the factors associated with morbidity and mortality from India.
- This study was planned with an aim to identify clinical and endoscopic profile of patients with UGIB coming to our hospital, which is a tertiary referral center and to study the factors associated with etiology, morbidity and mortality.

Aim of the Study

- To determine the common etiologies of upper gastrointestinal bleeding in patients presenting to GRH
- To evaluate variceal bleed as an initial presenting feature in chronic liver disease patients .
- To assess the risk factors associated with rebleed and mortality using scoring system.

Review of literature

Gastrointestinal (GI) bleeding is a common problem in emergency medical practice and should be considered potentially life threatening until proven otherwise.

Upper GI bleeding is defined as that originating proximal to the ligament of Treitz, whereas lower GI bleeding originates more distally. Clinically it is differentiated with the presenting symptoms and further investigations are done to confirm with simultaneous resuscitation and supportive treatment.

Ligament OF TREITZ is formed by a fold of peritoneum over the suspensory muscle of the duodenum. It is an anatomical landmark used to denote the duodenojejunal junction and the point where the small intestine passes from retroperitoneal duodenum to intraperitoneal jejunum. Clinicians use this as a reference to classify upper and lower gastrointestinal bleed.

Gastrointestinal bleeding can be occult or overt .Overt bleeding is manifested as hematemesis, melena or hematochezia.

Hematemesis is vomiting out of blood, bright red to coffee brown coloured .

Melena is passing dark coloured, tarry, foul smelling stools. Presence of melena indicates that the blood has been in the gastrointestinal tract for adequate time so as to be altered and converted to dark coloured stools. It is usually a minimum of 14 hours upto a period of 3 – 5 days. Melena requires a bleeding of around 50-60ml from the upper GI tract.

Hematochezia is passing bright red blood or maroon blood through rectum. This is usually a manifestation of lower GI bleed but it can occur due to bleeding from a proximal source when the transit time is short.

Epidemiology:

In current day practice , UGIB is one among the common medical emergency encountered. An overall annual incidence of Acute upper GI bleeding in adults is approximately 100 per 100,000. It is more common among males and markedly more common among the elderly. Its associated mortality rises with age. Lower GI bleeding is somewhat less common, with an annual incidence of approximately 20 per 100,000. This too, is more common among males and among the elderly.

Prevalence of UGIB in India and worldwide :

Endoscopic diagnosis	Deep Anand et al	Anand et al	Odisha	AAFP	Rathod et al	Jaka et al
	Dehradun North India	AIIMS, North India	Odisha, India	Geogia,US	Gujarat, India	Tanzania
Varices	56.14%	45.4%	12.8%	6%	24%	51.3%
PUD	14.91%	30%	57.6%	62%	22%	25.0%
Erosive lesions	12.28%	8.5%	1.8%	8%	34%	15.8%
Mallory-Weiss	8.77%		1.8%	4%	-	
Malignacy	4.38%		7.7%	2%	8%%	
Esophagitis				-	18%	1.7%%
Other	3.5%			2%		
Normal				8%		3.3%

AAPF(1) Deep Anand(2) Odisha(3)

Causes of upper GI bleed :

- Peptic ulcer disease
- Variceal bleeding
- Gastroduodenal erosive lesions
- Esophagitis
- Mallory weiss tear
- Neoplasm
- Vascular lesions

PEPTIC ULCER DISEASE:

Peptic ulcer disease are the most common causes of upper gastrointestinal bleeding . But epidemiological variation exists due to variation in food, lifestyle and economic pattern. Prevalence of PUD is ~ 12% in males and ~10 % in females.

Ulcer is the disruption in mucosal intergrity of stomach, duodenum.loss of mucosal surface more than 5 mm and depth upto submucosa defines the ulcer.

Pathophysiology :

The gastroduodenal system besides role in digestion , also performs immune defense and homeostasis of its own system. This is attained through gastrointestinal mucosa and epithelial and subepithelial components.

The gastroduodenal epithelium is exposed to constant insult to series of endogenous and exogenous substances; endogenous factors include hydrochloric acid, pepsin, pancreatic enzymes, bile salts and acids and exogenous substances include drugs, alcohol, microbes. The biologic system has its own defense to protect its mucosa from injury and the ability to repair any injury and the integrity is maintained by an intricate system. The protective mechanism includes,

i) **Mucus bicarbonate phospholipid layer** - acts as a physiochemical barrier to the effects of hydrogen ions and gastroduodenal enzymes. Mucus is produced from gastroduodenal surface epithelial cells, is primarily composed of 95% of water and a mixture of phospholipids and glycoproteins. This mucus layer acts as a non stirred layer of water impeding movements of molecules and ions. Bicarbonate which is also secreted by the same gastroduodenal epithelial cells into the mucus forms a pH gradient, which is 1-2 at luminal surface and 6-7 at the epithelial cell surface.

ii)Surface epithelial cells - which has the following functions

Mucus production,

Maintenance of pH,

Epithelial cell ionic transporters,

Bicarbonate production,

Intracellular tight junctions,

Heat shock proteins – prevent protein denaturation and protect cells from increased temperature , cytotoxic agents and oxidative stress.

Restitution – process of migration of gastric epithelial cells to a nearby injury and restoration of the damaged region to normal occur in surface epithelium. This requires uninterrupted blood supply and optimal pH (alkaline) at that site of lesion. This regeneration process requires presence of multiple growth factors and prostaglandins.

iii)Prostaglandin role in homeostasis of GI mucosa:

Prostaglandins play a vital role in gastric epithelial defense and repair. It effects control over the synthesis and release of bicarbonate secretion and mucus. It inhibit parietal cell secretion, and maintains blood flow to the mucosal layer and epithelial cell restitution.

Phospholipase A2 acts on the membrane phospholipids to form esterified arachidonic acid. Cyclo oxygenase (COX) is the enzyme which acts on arachidonic acid to form prostaglandins. COX plays the crucial step in the prostaglandin synthesis. There are two forms of isoforms – COX1 and COX2 each having its unique structure, distribution and function.COX1 is present in stomach, kidneys, platelet and endothelium; helps in maintaining the integrity of the organ in which it is expressed. COX2 is inducible by inflammation and expressed by macrophages,leukocytes, fibroblasts and synovial cells.

The role of Nonsteroidal anti-inflammatory drugs in the pathology of peptic ulcer disease is understood from its effect on COX and in the production of prostaglandlin. This explains the etiological role of NSAIDs in the Peptic ulcer disease.

Pathogenesis of PUD :

Definite risk factors in the development of PUD are

- *Helicobacter pylori*,
- NSAIDs,

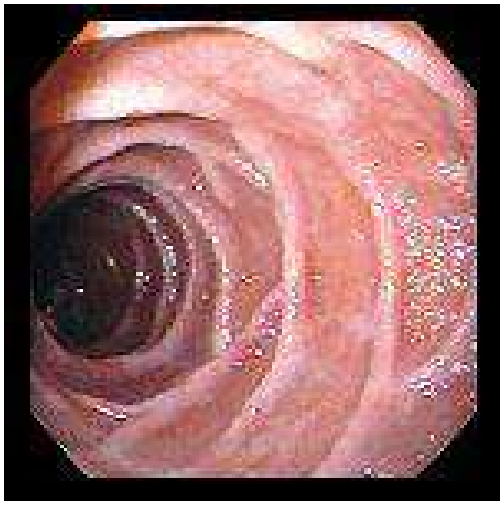
-these two are the most frequently associated risk factors.

Additional risk factors include

- chronic obstructive lung disease,
- chronic renal insufficiency,
- current tobacco use,
- former tobacco use,
- elderly age,
- coronary heart disease,
- former alcohol use,
- African-American race
- obesity and diabetes

Duodenal ulcer :

Most common site of duodenal ulcer is the first part of the duodenum. Around 90% of them are found within 3 cm of pylorus. The size of the ulcer varies from less than one centimetre to large as 6 cm. Ulcers of size 3 to 6cm are usually termed as giant ulcer. Ulcers have a sharply demarcated margin. Depth of the ulcer may reaches upto the muscularis propria. Histopathology features of the ulcer shows eosinophilic necrosis at the base with surrounding fibrosis. Duodenal ulcers are rarely malignant.



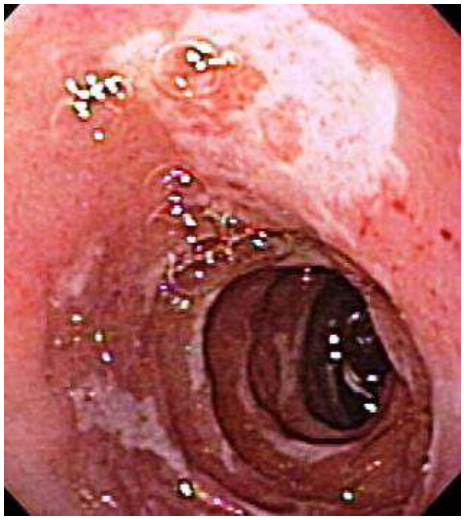


Fig.Normal duodenum Duodenal ulcer

Increased average basal and nocturnal gastric acid secretion and decreased bicarbonate secretion are the abnormalities associated with duodenal ulcer. *H.pylori* infection has been considered as the contributory factor to the foresaid abnormalities.

Gastric ulcer :

Gastric ulcers unlike that of duodenal ulcer can represent malignancy and biopsy should be done. Benign gastric ulcers are most often found distal to the junction between the antrum and the acid secretory mucosa and rarely occur in the gastric fundus.

Fig: Normal stomach Gastric ulcer

Histologically gastric and duodenal ulcers show similar features and that is the distribution and morphology which differentiates them .

H. Pylori infection is associated with benign gastric ulcer and shows antral gastritis. But gastric ulcers due to NSAIDS are not associated with coexixting gastritis. They may exhibit evidence of a chemical gastropathy - typified by foveolar hyperplasia, edema of the lamina propria, and epithelial regeneration . Extension of smooth-muscle fibers into the upper portions of the mucosa may also occur(4).

Gastric ulcers are associated with normal or decreased acid production .Mucosal defense may also be impaired. Gastric ulcers have certain characteristics based on their location.

Gastric ulcer classification and characteristics :

GASTRIC ULCER	SITE	ACID PRODUCTION
TYPE I	Body of stomach	Low
TYPE II	Antrum	Low to normal
TYPE III	Within 3cm of pylorus	Normal or High
TYPE IV	Cardia	Low

Helicobacter pylori :

H.pylori is a gram-negative microaerophilic rod with multiple sheathed flagella. It usually habitats in the deeper part of the mucosal layer and doesnot invade the cell under normal acidic environment. But it is capable of living within the aggressive environment of the stomach. Initially, *H. Pylori live*s in the antrum but move towards the more proximal part of the stomach.Under adverse condition, the organism transforms into a dormant state to survive.

The protective effect of acidic pH is tackled with the ability of the oraganism to change its surrounding into alkaline pH. This is done by the urease enzyme

activity of H.pylori which produce ammonia from urea and makes its environment alkaline.

The H.pylori infection differs among population and it depends on the quality of life of that region. Factors influencing infection with H.pylori are habitat in a developing nation i)congestion, ii)unhygienic living conditions,iii)contaminated water and food, and iv)exposure to gastric contents infected with *H.pylori*

Human to human transmission occurs through oral-oral or faecal- oral route.

NSAIDs:

Side effects of nausea and dyspepsia occurs in around

50–60% and peptic ulceration occurs in ~15–30% of individuals taking NSAIDs regularly. Complications of PUD occur in about ~1.5% individuals among them.

The role of NSAIDs in the pathology of peptic ulcer disease is understood from its effect on COX and in the production of prostaglandlin. The beneficial therapeutic effect of NSAIDs occur due its inhibitory effect on COX2 and the toxicity is due to its inhibitory effect on COX1 isoform leading to mucosal ulceration and renal dysfunction. COX2 selective inhibitors have advantage over nonselective NSAIDs in minimising the tocic effects on mucosa. But certain drugs cause adverse effects on cardiovascular system and

myocardial infarction.This explains the etiological role of NSAIDs in the Peptic ulcer disease.

Most of the patients who develop serious NSAIDs related complications do not experience preceeding dyspeptic symptoms. In this regard, NSAIDs related complication should be borne in mind in asymptomatic patients with chronic drug intake. Concomitant *H. pylori* infection increases the risk of PUD-associated GI bleeding in chronic users of low- dose aspirin. Established risk factors for NSAIDs induced PUD include advanced age, history of ulcer, concomitant use of glucocorticoids, high-dose NSAIDs, concomitant use of anticoagulants, clopidogrel, multisystem disease or organ failure, cigarette smoking, and alcohol consumption.

Smoking :

Smoking is a risk factor for peptic ulcer disease and following mechanisms has been ascribed.

Decreased bicarbonate production

Altered gastric emptying,

Generation of noxious mucosal free radicals.

VARICES :

Varices are important cause for upper gastrointestinal bleeding. Varices reflects the presence of portal hypertension. Portal hypertension is defined as elevated hepatic venous portal gradient more than 5 mmHg.

Gastroesophageal varices develop in 5–15% of cirrhotics per year.Most of the cirrhotic patients will develop gastroesophageal varices with time. The apperance of gastroesophageal varices shows relation with the severity of liver disease. Varices in present in 40 percent of Cirrhotics with child pugh score A , and 85 percent of child pugh score C patients(5).

Anatomy and physiology of Portal venous system :

The portal vein is formed by the confluence of the superior mesenteric and splenic veins.

Spleenic vein drains blood from the spleen, fundus of stomach and part of the pancreas. Inferior mesenteric vein drains blood from the large intestine - transverse colon,descending colon and rectum – superior two thirds usually joins the spleenic vein

Superior mesenteric veins drain blood from small intestine, large intestine, stomach , pancreas and appendix.

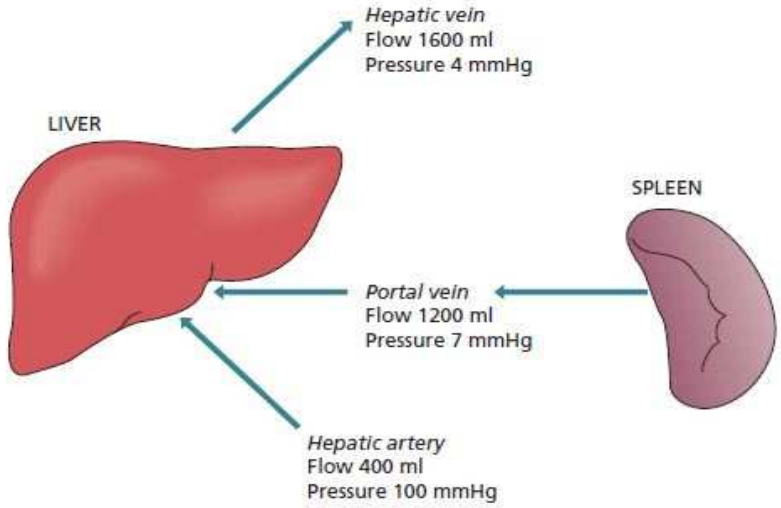


Fig.Normal portal venous flow and pressure

Portal hypertension leads to two major complications – variceal hemorrhage and ascites.

Pathophysiology of varices formation :

Anatomically collaterals usually exists between the portal venous system and the systemic venous system at certain anatomic locations. Blood flows from the systemic circulation into the portal system.

Two pathogenesis contributing to portal hypertension are

- Increased resistance to portal venous flow due to extrahepatic obstruction or intrahepatic resistance due to cirrhosis and regenerative nodules.

- Increased splanchic blood flow due to vasodilation of splanchic circulation.

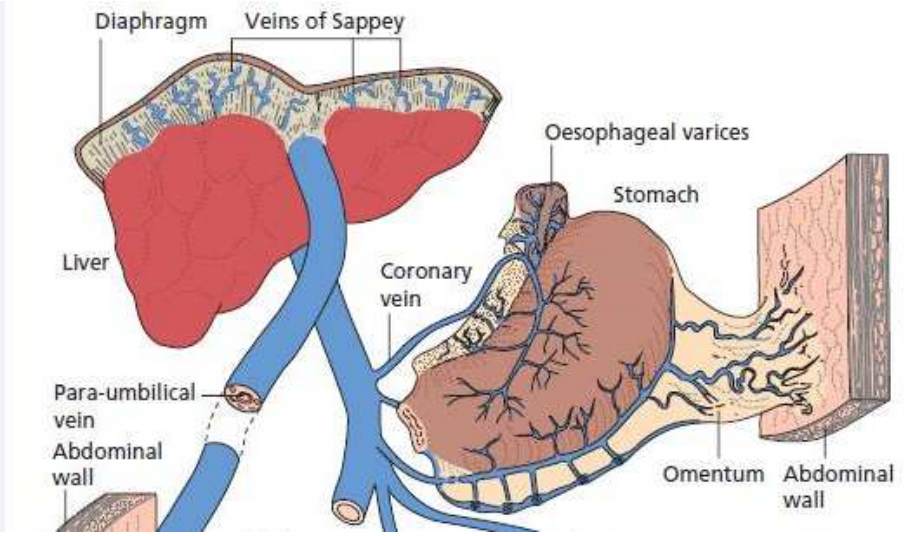


Fig.Collaterals formation at esophagus in portal hypertension

When portal hypertension develops, the resistance in the portal vessels pressure raises more than that of the systemic system,and causes reversal of flow . This is transmitted to the portosystemic junctions and the collaterals become dilated and distended

Angiogenesis and new blood vessel formation also occur with an effort to increase collataeral bed to decompress the portal hypertension but when the collateral is unable to withstand the pressure, dilation of vessels occur.further compromise leads to complication of rupture and bleeding.

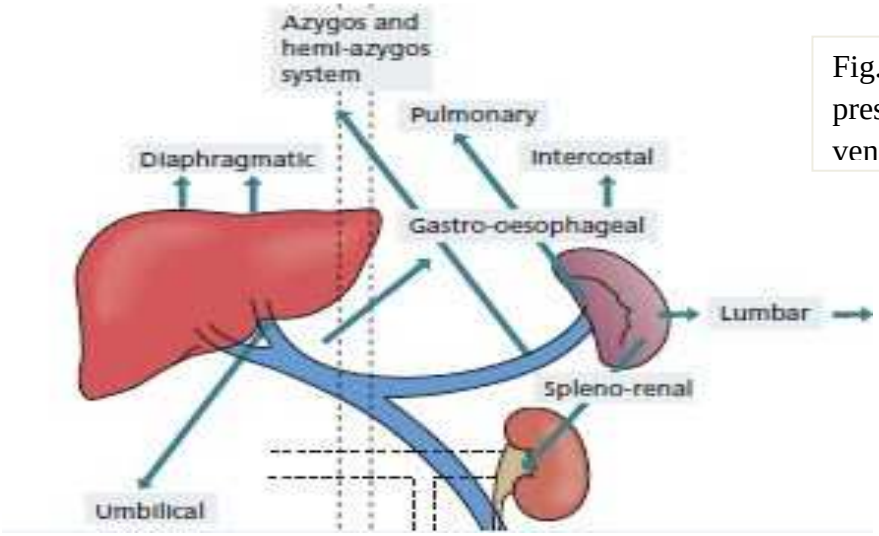


Fig.Direction of flow and pressure in intrahepatic portal venous obstruction

Varices should be kept in mind while treating all patients with clinical picture of liver cell failure. Once diagnosis of cirrhosis is made, the patient should be subjected to endoscopy to rule out varices and appropriate management should be done. When absent, periodic review and yearly endoscopy should be done

Anatomical location of varices in UGIB:

Causes of portal hypertension

1.Prehepatic causes

Portal vein obstruction

- Idiopathic
- Cirrhosis
- Infection
- Pancreatitis

- Abdominal trauma.
- Coagulation disorders
    - polycythemia vera,
    - essential thrombocytosis,
    - deficiencies in protein C,
    - protein S deficiency,
    - antithrombin 3 deficiency,
    - factor V Leiden

Spleenic vein thrombosis

Massive spleenomegaly – Banti's syndrome

2.Hepatic causes :

Presinusoidal

- Schistosomiasis
- Congenital hepatic fibrosis

Sinusoidal

- Cirrhosis of any etiology
- Alcoholic hepatitis

Postsinusoidal

Hepatic sinusoidal obstruction

3.Post hepatic causes

- Budd-Chiari syndrome
- Inferior vena caval webs
- Cardiac causes
- Restrictive cardiomyopathy
- Constrictive pericarditis
- Severe congestive heart failure

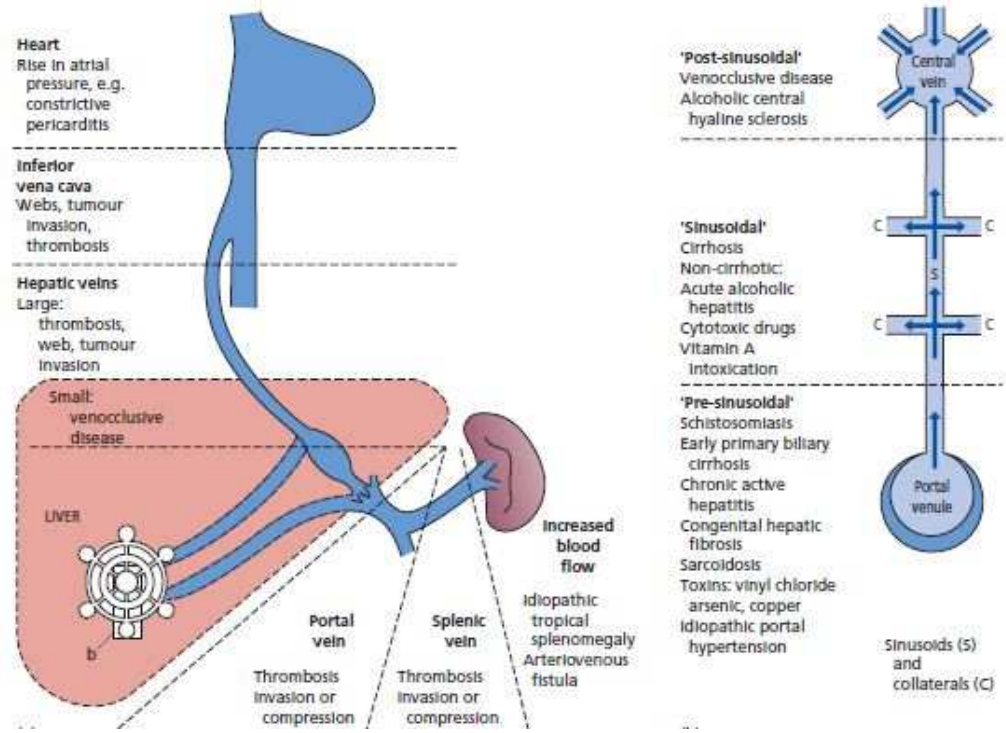


Fig.Causes of portal hypertension

Esophageal varices:

Gastroesophageal collateral bed is the common site of variceal formation and bleeding. When the HVPG exceeds 10 mm Hg, esophageal varices develops. In esophagus, the varices along the lower 2 to 3 cm submucosa lies very superficial, have fragile thin wall and so why bleeding usually occurs at this site. These vessels do not communicate with the periesophageal veins and therefore cannot easily be decompressed(8)*.

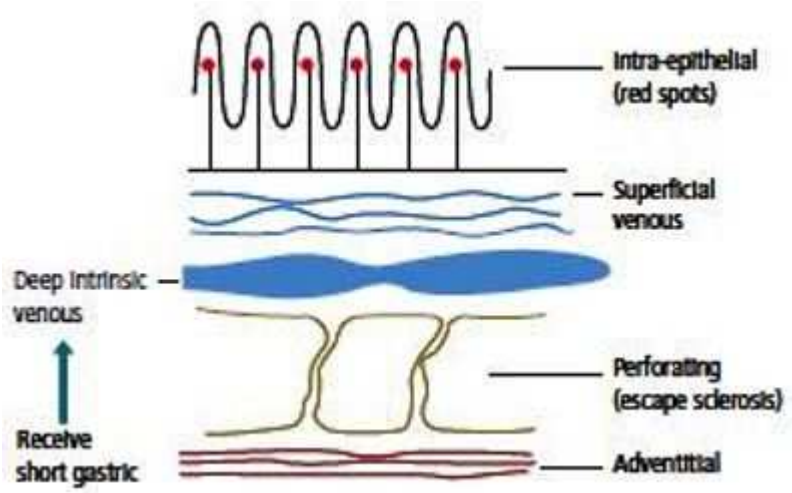


Fig:Esophagus - subcutaneous venous plexus

Esophageal varices may be small – less than 5mm or large –greater than 5 mm.Small varices progress with time into large varices. The predictors of first bleeding include the size of varices, severity of cirrhosis (Child B or C), variceal pressure (>12 mm Hg), and the endoscopic presence of red wale marks(5)

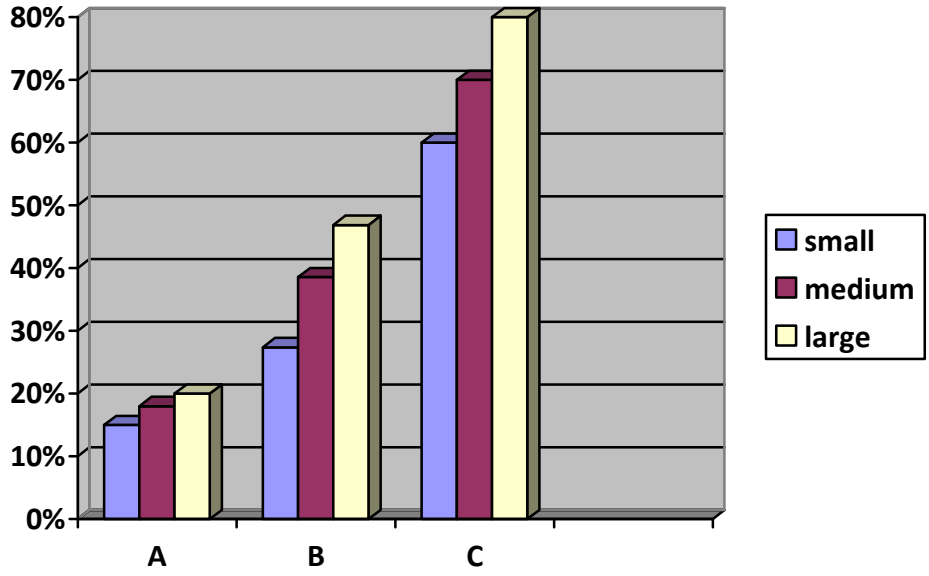


Fig: Probability of bleeding with relation to size of varices and Child pugh score

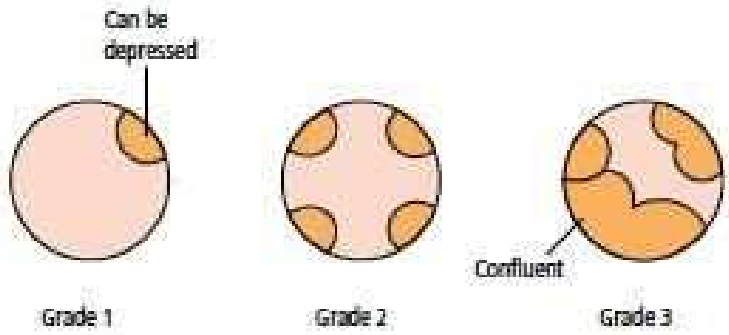


Fig:Endoscopy Grading of Esophageal varices

CHILD PUGH Score: (6)

Parameter	Score 1	Score 2	Score 3
Ascites	None	Mild/Moderate	Tense
Hepatic encephalopathy	None	Grade 1-2	Grade 3-4
Bilirubin (mg/dL)	<2	2-3	>3
Albumin g/L (g/dL)	>3.5	2.8-3.5	<2.8
PT (Sec over control)or	<4	4-6	>6
INR	<1.7	1.7-2.3	>2.3

Childpugh A 5-6; B 7-9; C 10-15

Gastric varices :

Gastric varices occur less common than esophageal varices around 5 – 30% (5) of portal hypertension. Bleeding occurs in about one-fourth of them. Gastric varices can occur in isolation or as extension of esophageal varices.

Gastic varices are classified according to the site and association with esophageal varices.(*Sarrin et al*.)

Gastroesophageal varices[2]

- Type 1 varices - extend along the lesser curvature.
- Type 2 varices - extend along the fundus. They are longer and more tortuous than GOV1

Isolated Gastric varices [2] :

- Type 1 - fundus and tend to be tortuous and complex
- Type 2 - body, antrum, or around the pylorus.

Isolated gastric varices at fundus may occur due to spleenic vein thrombosis and should be ruled out.

Ectopic varices:

Varices may occur anywhere in the gastrointestinal tract.when present in GI tract other than esophagus and stomach, they are termed as ectopic varices. They are difficult to identify and continue be a concealed source of bleeding .once identified the appropriate treatment modalities are not well established.

Management

Endoscopy has evolved as the principal diagnostic as well as therapeutic tool in management of upper gastrointestinal bleed. Endoscopy era has made to review the etiology of upper GI bleed as the most common cause varies with region. It helps in identifying individuals and categorising them accordingly to be managed by primary prophylaxis in newly diagnosed cirrhotic or need for endoscopic intervention or surgery.

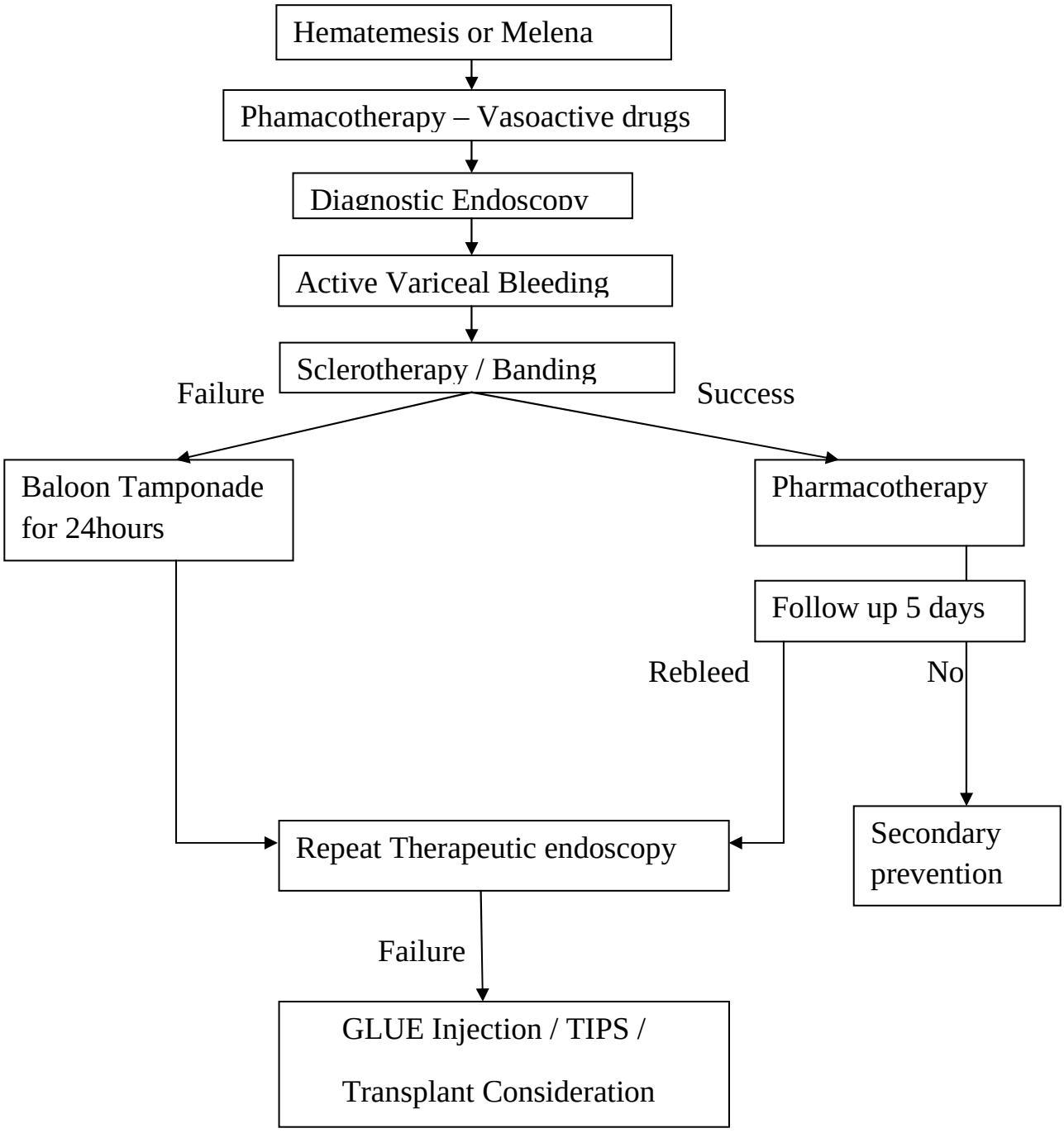


Fig: Management of Acute variceal bleed

Patients presenting with upper GI bleed should be stabilised before endoscopic intervention. This include insertion of nasogastric tube to confirm as well as quantify the amount of blood loss, maintaing intravascular volume by crystalloids, colloids. Blood transfusion is done when patient has anaemia.once the patient was stabilised,endoscopy is planned as early possible preferentially within 24 hours. Though advancement in technique and treatment has occurred, the availability of the endoscopy facility determines the timing of endoscopy.

MALLORY-WEISS tears :

Mallory – weiss tear is a nontransmural tear at the gastroesophageal junction that is caused by vomiting, retching, or vigorous coughing. It is one among a common cause of upper gastrointestinal bleeding. Most patients present with hematemesis but antecedent vomiting is not always evident as that of expected. Bleeding usually stops spontaneously, but protracted bleeding may occur. The prolonged bleeding is managed by injection of local epinephrine or cauterization therapy, endoscopic clipping, or angiographic embolization. Surgery is rarely needed.

Erosive Gastroduodenal disease :

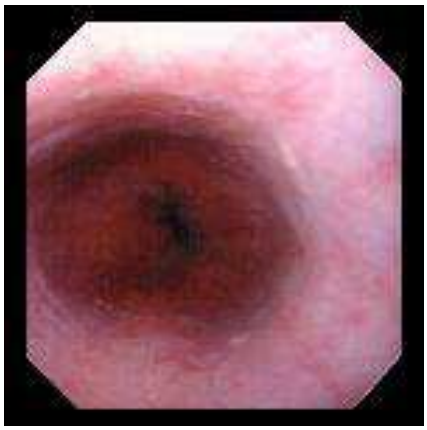
Acute erosive gastric mucosal lesions or frank ulceration with bleeding can be precipitated by any form of stress. This includes sepsis, burns, severe injury, shock, head injury etc and manifest usually 48-72 hours after the insult.

Fig: Erosive gastric mucosal lesions

These stress induced ulcers are most commonly found in the acid-producing portions of the stomach – body and fundus of stomach. They manifestation usually as abdominal discomfort associated with acute gastrointestinal bleeding. The severity of bleeding is minimal but may be life threatening at times. Patient under mechanical ventilation and underlying coagulopathy are risk factors for bleeding.

Esophagitis :

Esophagitis is the inflammation of esophagus due to various etiologies.



Normal esophagus

Esophagitis

Lax lower esophageal sphincter leads to gastroesophageal reflux disease which is the common cause. Risk factors include Alcohol consumption, smoking, medication – NSAIDs. Pill esophagitis, eosiniophilic esophagitis, infections due to H.pylori, Candidiasis, Radiation and corrosive induced lesions are other causes.

Vascular Lesions :

Vascular ectasias are flat mucosal vascular anomalies that usually causes slow intestinal blood loss. This occurs as either in a sporadic fashion or in a

well-defined pattern of distribution. e.g., gastric antral vascular ectasia - GAVE or “watermelon stomach” .

They are often responsive to local endoscopic ablative therapy, such as argon plasma coagulation.

Patients with diffuse ectasias associated with chronic renal failure and with hereditary hemorrhagic telangiectasi, the chances of recurrent bleed occurs despite endoscopic treatment of easily accessible lesions by conventional endoscopy. These patients may benefit from deep enteroscopy with endoscopic therapy, pharmacologic treatment with octreotide or estrogen/progesterone therapy, or intraoperative enteroscopy.

Laboratory investigations in Acute GI bleed:

Laboratory data assists in the management of acute GI bleed. Haemoglobin does not fall rapidly unless there is severe bleed. Fall in Hematocrit helps in assessment of severity of blood loss. The change in hematocrit occurs after about 24 hours and the delay is due to proportionate loss of plasma along with erythrocytes. The subsequent fall in dilution of hematocrit is due to movement of extravascular fluid into vascular compartment. This may be further altered by fluid resuscitation.

Thus significance of a single hematocrit value cannot be inferred unless a recent previous report is available. Series of hematocrit values are done to monitor the status of patient and this should be correlated clinically and according to the fluid administered. More accurate assessment of volume status can be done with central intravenous catheter and applied when it is highly useful in patients with co existing renal or cardiac failure.

Investigations done towards etiology includes monitoring of liver and renal parameters, coagulation profile, white blood cell count, platelet count. Anaemia is normocytic normochromic in acute GI bleed and becomes microcytic in chronic loss due to iron deficiency. Leukocytosis occur due to stress, but infection should be ruled out. Thrombocytopenia reflects hypersplenism due to portal hypertension. Thrombocytopenia is an early indicator of onset of portal

hypertension. Renal parameters especially urea is elevated as a result of hypovolemia and also due to the absorption of denatured proteins in the blood that has passed through the intestine.Elevated urea creatinine ratio indicates the prerenal azotemia due to hypovolemia.

Management of Upper GI Bleed :

General measures :

Nasogastric tube :

Nasogastric aspiration with saline lavage should be done routinely to detect the presence of intragastric blood, to assess the bleed quantitatively, and clear the gastric contents to prevent aspiration as well a prerequisite in case of emergency endoscopy management.. A gross bloody aspirate confirms a UGIB, unless trauma occurs during nasogastric tube insertion. Bright red blood suggests currently active bleeding, whereas coffee grounds suggest recently active bleeding. Absence of bloody aspirate doesnot exclude upper GI bleed, since gastric contents may have emptied during the transit period.

When nasogastric aspiration is impossible prior endoscopy, erythromycin . Intravenous infusion of 250 mg ~ 30 min helps in rapid gastric

emptying.emptying gastric contents helps in better endoscopic visualisation and avoids repeating the procedure.

Ice cold saline/water instillation through ryles tube causes local vasoconstriction and reduces bleeding temporarily .

Blood transfusion :

Blood transfusion, fresh frozen plasma are transfused appropriately

Pharmacotherapy :

NonVariceal Bleed :

- Protein pump inhibitors patenteral
- Antibiotics
- Anti – H.Pylori regime

Variceal Bleed :

- Proton pump inhibitors
- Prophylactic Antibiotics –parenteral
- Vasoactive drugs

- Vasopressin / Terlipressin
- Stomatostatin and analogues
-

Proton pump inhibitors :

Proton pump inhibitors acts by inhiting gastric proton pump(H+K+ATPase).These are prodrugs that require acidic medium for activation. Once absorbed into circulation these drugs reach the parietal cells of stomach and gets attached to canaliculi.The binding to canaliculi is irreversible and makes the duration of action longer until new pumps are formed.Since these drugs block the pumps, they are more effective.

These patients requiring immediate acid suppression are treated with parenteral pantoprazole or lansoprazole. A single intravenous bolus of 80 mg of pantoprazole inhibits acid production by 80% to 90% within an hour and this inhibition persists for long duration, permitting once daily dosing.

Since most causes of upper GI bleed are associated with acid secreting abnormalities and mucosal injury like gastric ulcer, duodenal ulcer, esophagitis, erosive mucosal lesions proton pump inhibitors blocks the common pathogenesis involved. This reduces the severity and further active bleed.

Vasoactive drugs :

Vasopressin and terlipressin causes vasoconstriction of splanchic arterioles and decreases the in flow of blood and lowers portal pressure. Vasoconstriction at other organs may cause colicky or myocardial ischemia.

Dose of *vasopressin* is 2 mg i.v. QID for 48 h.Further dose of 1 mg every 4 – 6 h may be continued for a further 3 days.

Stomatostatin and octreotide decreases blood flow to the gastrointestinal tract andt has been used to treat bleeding esophageal varices, peptic ulcers, and postprandial orthostatic hypotension. Standardly used to reduce the risk of bleeding from esophageal varices because it inhibits mesenteric vasodilatation induced by glucagon.

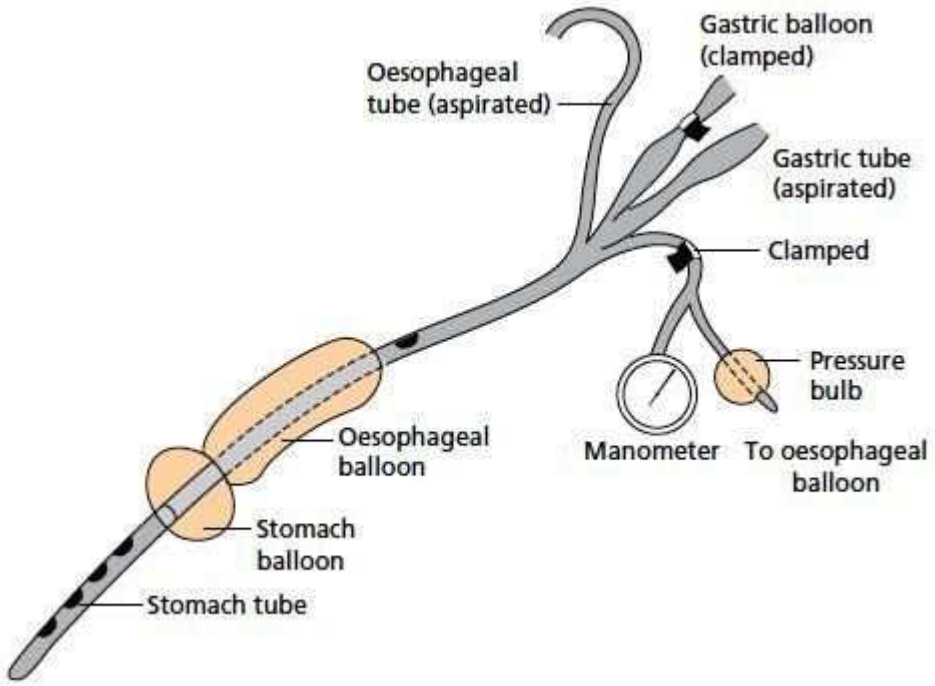
Stomatostatin 250microgram iv infusion followed by 6mg/kg infusion over 24hours. Treatment is initiated in suspected cases of variceal bleed, previous history of varices, clinical evidence of liver disease and elevated biochemical parameters.

Octreotide is administered at dose of 50 -100 microgram / hour infusion.

Sengstaken-Blakemore tube :

Esophageal balloon compression causes occlusion of submucosal vessels and stops bleeding. Sengstaken-Blakemore tube consists of esophageal and gastric balloon. The gastric balloon is inflated first to place the tube in position. The pressure applied at esophageal balloon shoulb be greater than the expected portal pressure. Once both balloons are inflated slight traction is

applied to avoid the tube from slipping into stomach. After 12 hours esophageal balloon is deflated and check for bleeding. If recurs reinflate till 24 hours.



This procedure used as a temporary method to control bleeding, during transportation to referral center or till dfinite therapy is available. Compression should not be given more than 24hours.

ENDOSCOPY :

It is the initial test in the evaluation of Upper gastrointestinal bleed. Upper GI Endoscopy is the technique through which gut lumen is visualised through a scope passed through oral cavity.The modern era of endoscopy began

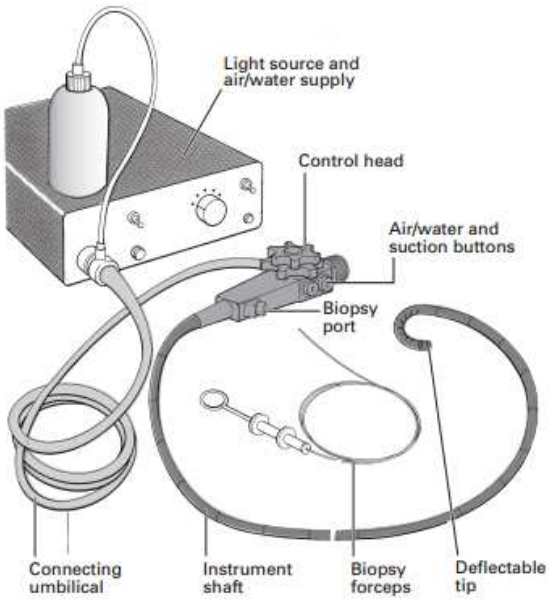
with the development of fibreoptic instruments in the 1960s. For most purposes these are being supplanted by video chip endoscopes in the 1990s(8)

It is most essential tool in evaluation of suspected peptic ulcer disease, neoplasm, malignancy, esophagitis, unexplained anemia, malabsorption. Patient presenting with chronic symptoms of dyspepsia, vomiting, unintentional weight loss, anaemia, malabsorption are benefitted by endoscopic evaluation. Scopy besides identifying the lesion also provides the opportunity to confirm the suspected lesion by access to biopsy in the first sitting when present.

Endoscopy offers the advantage of therapeutic intervention in single sitting. The endoscopic techniques have evolved over time with developing technologies and include sclerotherapy,injection of adrenaline, variceal ligature banding, glue injection,endoscopic clippings, electrocautery and argonplasma anticoagulation.

Instrument [1] :

They basically consists of i)control head ii)flexible shaft. The shaft is designed technically in such a way that it’s tip can be manipulated with controls at the head. Umbilical cord is the part through which light source reaches the head. It serves as a link through wich external light, air, suction and water is connected to the control head. The suction channel is used for the passage of diagnostic tools (e.g. biopsy forceps) and therapeutic devices



Fibrooptic instruments :

The fibro-optic cable bundle is 2–3·mm in diameter and contains 20·000–40·000 fine glass fibres, each close to 10·μm in diameter. Each fibre carries the light focussed on it to the other end of the cable by using the property of repeated internal reflections of light. The light transmitted to other end is processed to form the image based upon the spatial orientation of the individual fibres throughout the shaft. Leakage of fibre from each fibre is achieved by coating each fibre with glass of lower optical density. Thus the flexible endoscopy is designed technically that it can transmit image even when it is flexed to make a knot.

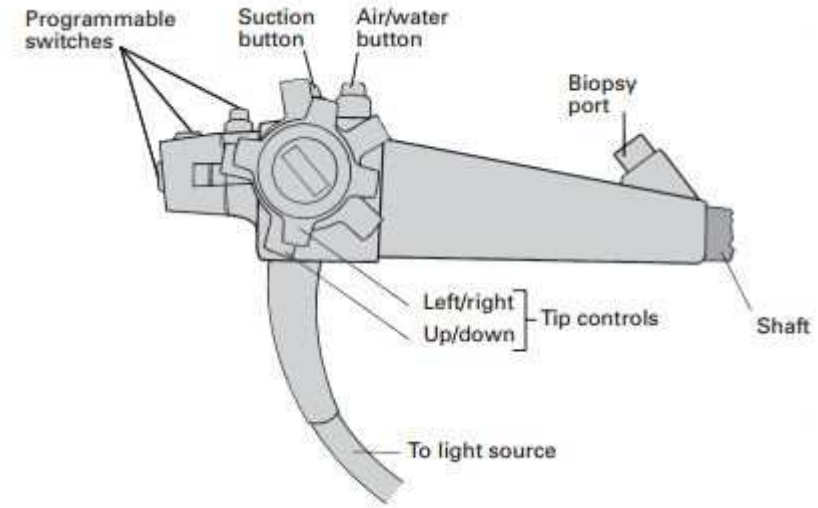
Videoendoscope[1] :

Videoendoscope uses a chip (charged couple device -CCD)mounted at the tip of the scope which absorbs light from the image. Instead of fibreopticals, to and fro wiring connect the chip to the control head by way of electronics. The site of the ocular lens in a fibre-endoscope is replaced by controls and knobs in videoendoscope to focus on a lesion. This allow the scopist to have a view at a screen and the instrument can be manipulated via the control head.

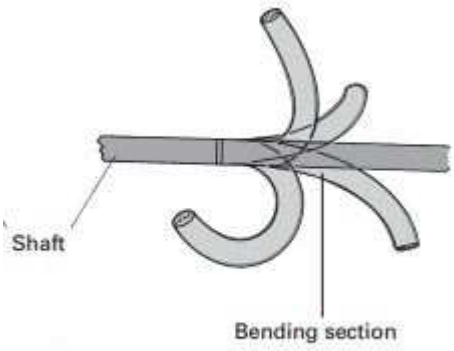
Videoendoscopes offer the advantage the gut can be visualised by everyone in the room, which is restricted only to the scopist in case of fibroscope. It also provides better clarity in the printed copy of the obtained image.

Light source – is obtained from an external high beam source which is transmitted through the umbilical cord to the scope.

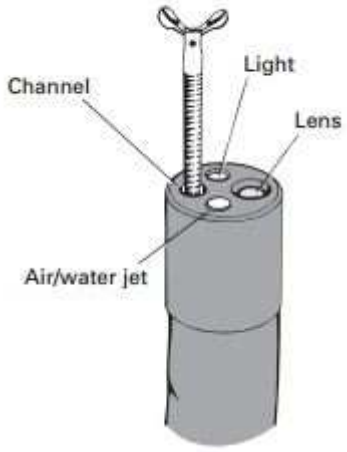
Instrument control :



The instrument tip is manoeuvred with help of wires attached just beneath the tip, which passes through whole length of the scope. These wires are controlled with provisions at the control head. The tip can be moved right, left, up or down and can be fixed at any angulation. This causes no damage to the instrument.



In addition to the visual imaging, the diagnostic and therapeutic manoeuvres are achieved via the provisions for instrumentation through the shaft. Instrument accessories are made to pass through the shaft via ports at the control end, to the



field of view.

The following flexible instruments are passed through,

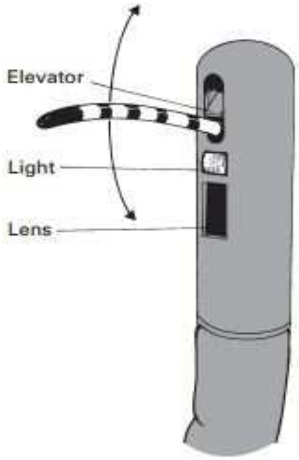
Biopsy forceps

Cytology brush

Diathermy

Sclerotherapy needle

Detachable snare



Irrigation system with suctioning facilitates flushing the lesion, identifying oozing point. Air can be insufflated through the shaft. The water and air supply is obtained from external source via the umbilical cord to control head.

Upper GI scopy passes through esophagus, stomach, upto first and second part of duodenum. It is the initial test in the evaluation of suspected peptic ulcer disease, neoplasm, malignancy, esophagitis, unexplained anemia, malabsorption. Patient presenting with chronic symptoms of dyspepsia, vomiting, unintentional weight loss, anaemia, malabsorption are benefitted by endoscopic evaluation. Scopy besides identifying the lesion also provides the opportunity to confirm the suspected lesion by access to biopsy in the first sitting when present.

Indication for upper GI scopy in suspected UGIB :

Hematemesis

Melena

Hematochezia

Anemia

Suspected Malignancy

Peptic ulcer disease

Unintentional weight loss

Dyspepsia with signs of organic disease

Indications for endoscopic therapy(9):

- Active spurting or oozing bleeding or a non-bleeding visible vessel
- Adherent clot resistant to vigorous irrigation

Ulcer with a clean base or a flat pigmented spot needs no endoscopic intervention.

Treatment with proton pump inhibitors should be continued with via parenteral route for patients who have stigmata of recent hemorrhage like oozing from vessel, adherent clot, non bleeding visible vessel. oral dosage of pantaprazole or other PPIs should be continued for findings of clean base ulcer or pigmented spots.

Repeat procedure is done when there is recuuence of bleed after initial endoscopic management. When further episodes occur surgery or arterial embolisation should be considered.

Stigmata of hemorrhage in ulcer :

FORREST CLASSIFICATION of Stigmata of Hemorrhage

Stigmata of hemorrhage	Forrest classification		Prevalence
Active spurting bleed	IA		12%
Active oozing bleed	IB		
Non-bleeding visible vessel	IIA		8%
Adherent clot	IIB		8%
Flat pigmented spot	IIC		16%
Clean Base	III		55%

ROCKALL SCORE :

For scoring 0, 1, or 2 rebleeding occurs in less than 5% of patients and mortality is virtually zero whether rebleeding occurs or not. The scoring system can be used to identify the one quarter of patients who are at negligible risk of dying but this can only be done only after confirming the diagnosis by endoscopy for stigmata of recent haemorrhage. An admission Rockall score of zero is often cited as identifying low risk patients, Patients with a Rockall score of ≤ 2 are generally accepted as being at low-risk of poor outcome, As with all true emergencies, the traditional triad of medical history, physical examination, and diagnosis often must be accomplished simultaneously with resuscitation and stabilization. Rebleeding has its most profound influence on mortality in the middle risk groups that score 3 or 4, when it is associated with an

approximately fivefold increase in mortality. In risk groups 5 to 7 rebleeding is associated with an approximately threefold increase in mortality and for risk group 8, a twofold increase(10) Rockall score also serves as prognosis both before and after a definitive endoscopic diagnosis. It helps in prioritising patients to be admitted to undergo endoscopy where there is limited availability of facilities and human resource.

Based on the score, it helps whether to treat patient as inpatient or period of stay based on the stigmata of recent hemorrhage.

GLASGOW BLATCHFORD SCORE(11) :

SCORE	RISK STRATIFICATION
0	Low risk Reasonable to manage as out patient
1 – 5	Increased risk for intervention Inpatient treatment is recommended Most patients recover without significant intervention
>5	High risk for intervention

GBS of zero has been reported to have > 99% sensitivity in identification of those who do not require intervention, rebleed or die in studies from Hong Kong (China)[30], United States[31], Japan[38], Taiwan (China)[27] and United Kingdom[18,23,25,28]. low risk patients suitable for out-patient management to those with GBS ≤ 1 or ≤ 2, but safety of this approach requires further study

Endoscopic procedures

Endoscopic injection sclerotherapy (EST) [2]

More than half a century after it was introduced by Crafoord and Frenckner [20], EST remains the primary treatment for bleeding esophagogastric varices in many parts of the world. It is used both in the control of acute bleeding and in elective obliteration of varices. Autopsy studies indicate that venous thrombosis, mucosal ulceration and acute inflammatory reactions can be found in the injection site as early as 2 days after sclerotherapy (13)

1.Intravariceal injection - Injections may be injected into the veins or

2.Paravariceal injection – injected into esophageal wall adjacent to the varices.

The intravesical injection is usually preferred. Sclerosant should be injected into each variceal column starting from the lower esophageal gastric junction upwards to the mid esophagus.

Sclerosing agents are two types,

i) fatty acids ii)dehydrating agents

Fatty acids- e.g. sodium morrhuate, ethanolamine oleate, polidocanol or
Dehydrating agents - e.g. sodium tetradecyl sulfate, ethanol, hypertonic glucose, and phenol.

1–2 ml are injected into each column at esophagogastric junction. A total volume of 20 ml can be injected at each session [2]

Post sclerotherapy :

Patients are observed for several hours and if no bleeding occurs, oral intake is resumed. Acid suppressive therapy and mucosal protectives are used to prevent excessive ulceration of esophagus. Patient is reviewed biweekly or more for futher sittings to make the remnant varices sclerosed.

Endoscopic variceal ligation :

Endoscopic variceal ligation was first introduced by Stiegmann and Goff in 1986 [28,29]. Variceal banding involves strangulation of varices by elastic circular ligatures. These ligatures are mounted on tip of endoscope. The varix is

sucked into the scope completely and the ligature is released, such that it constricts the neck of the scucked in part. Banding should begin distally and moved spirally towards proximal esophagus since the ligated varix obstructs vision and passage of the scope.This approach prevents the chance of obstruction of food passing through esophagus in later period.

Multi fire devices has overtook the initial single fire devices avoiding the need for passing the scope multiple times in case of multiple varices.

EVL obliterates varices by causing necrosis of mucosa and submucosa of the esophageal wall. After few sitting of EVL, the varices become small and the esophagus is scarred.

Demerits :

Early recurrence of varices is a problem associated with EVL. Esophageal varices may recur within months after treatment and it is more frequent in EVL. Recurrence is considered due to persistence of perforating veins in esophagus.These veins are considered as the determinant factor for recurrence; are not occluded by EVL. In almost all studies comparing the long-term effects of EST vs. EVL, recurrence of esophageal varices were found more frequently in the EVL-treated patients (30–48%) compared to the EST-treated patients (8–30%)(14)(15)

Rebleeding from gastric varices may be more common among those treated by EST than by EVL (37% vs. 8%)[2] The overall survival rates are similar in both treatment modalities and it is determined by hepatic reserve rather than by the method of endoscopic hemostasis.

Cyanoacrylate :

Endoscopic injection of the varices with N-butyl-2-cyanoacrylate was first introduced by Soehendra in 1995(16). The tissue adhesive coagulates almost instantaneously when in contact with blood. It is mixed with lipoidal solution to prevent adhesion in the scope channel. The procedure is carried out under fluoroscopy surveillance to ensure injection into the dilated varices.

The injected submucosal vessels become hard and the bleeding is stopped. Later the vein sloughs off and fall.Cyanoacrylate is very effective in controlling active spuring variceal haemorrhage and achieve acute hemostasis.

It is the preferred treatment modality in gastric varices where banding is not possible.

Complications :

Esophageal ulceration,Stricture,Spasm,Septic and organ emboli in cyanoacrylate,Peritionitis,Bacteremia

Detachable snare :

Varices is sucked into the scope and a detechable snare is released to enclose it. The varices then appear as a polyp with a string attached to its base. The varices gets thrombosed and falls off. Advantage is does not require multiple sitting, but needs technical expertise.

Treatment of H.pylori :

Triple regimen

PPI tablets bd Clarithromycin 500mg bd Amoxicillin 1 g bd

PPI tablets bd Clarithromycin 500mg bd Metronidazole 500mg bd

Quadraple regimen :

Omeprazole (lansoprazole) 20 mg bid (30 mg bid) Tetracycline 500 mg tid Metronidazole 500 mg tid Bismuth salicylate two tab qid

Study method and material

Study population:

This study was conducted among 100 patients who presented with upper GI bleed and underwent upper GI scopy at Government Rajaji Hospital, Madurai, during the study period.

Inclusion criteria:

1.Patients undergoing upper GI endoscopy for evaluation of upper GI bleed comprising hematemesis or/and melena.

2.Age > 14years

3.Gender: Both Male and Female

Exclusion criteria:

Age < 14 years

Known case of bleeding diathesis

Patients who satisfy inclusion criteria and are hemodynamically unstable to undergo upper gastrointestinal scopy

Design of study:

Descriptive Observational study

Period of study:

Six months January 2016 to July 2016

Ethical clearance:

Necessary ethical clearance was obtained from ethical committee ,GRH , Madurai.

Consent: Individual written and informed consent.

Analysis: Statistical analysis.

Conflict of interest: Nil

Data collection:

- Patients who presented with upper GI bleed were assessed clinically and evaluated . Supportive measures and treatment were initiated and once patient are hemodynamically stable, endoscopy is done after obtaining consent. Clinical parameters, Laboratory values, Endoscopic finding are assessed and documented and appropriate therapy was provided. Patients were observed post procedure and treated as inpatient or outpatient based on their health status. Follow up and review was done regularly.

Statistical analysis :

The obtained data were recorded and analysed. Statistical analysis of frequencies, percentage, mean, chi-square test were done using computing system with the recent available tool – IBM SPSS statistics version 21. Statistical values were interpreted and significance recorded and results were reported.

Results and analysis of the study

Table 1: Age distribution among study population:

Age in years	Frequency	Percentage %
<20	2	2
21-30	12	12
31-40	23	23
41-50	29	29
51-60	25	25
61-70	7	7
>70	2	2
Total	100	100

In this Study, total samples of 100 patients, most of them falls under 4th and 5th decade. Age 40 -50 years comprises 29%; 50-60 years comprises 25% and 30-40years comprise 23%; 20-30 years comprises 12%.

Age Distribution among study population

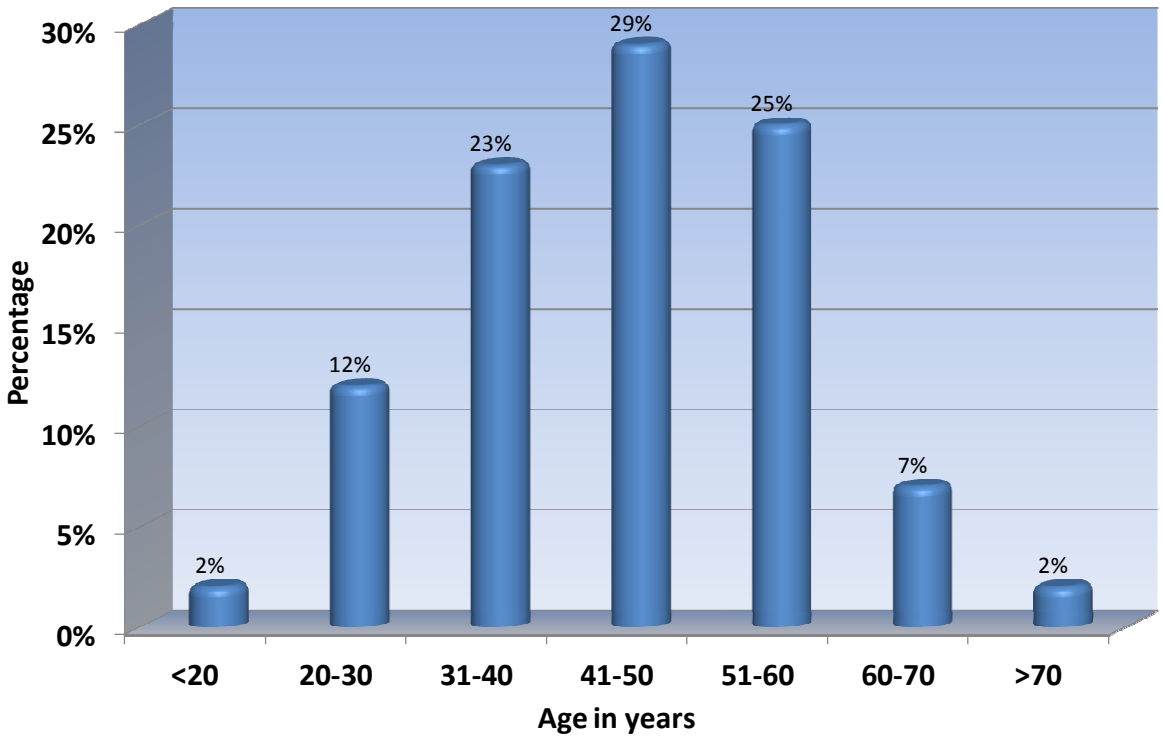


Table2 : Gender distribution among the study population :

Sex	Frequency	Percent
Male	72	72.0
Female	28	28.0
Total	100	100.0

Sex distribution shows more of males contributing 72% and females 28%. This reflects more prevalence of upper GI bleed in males.

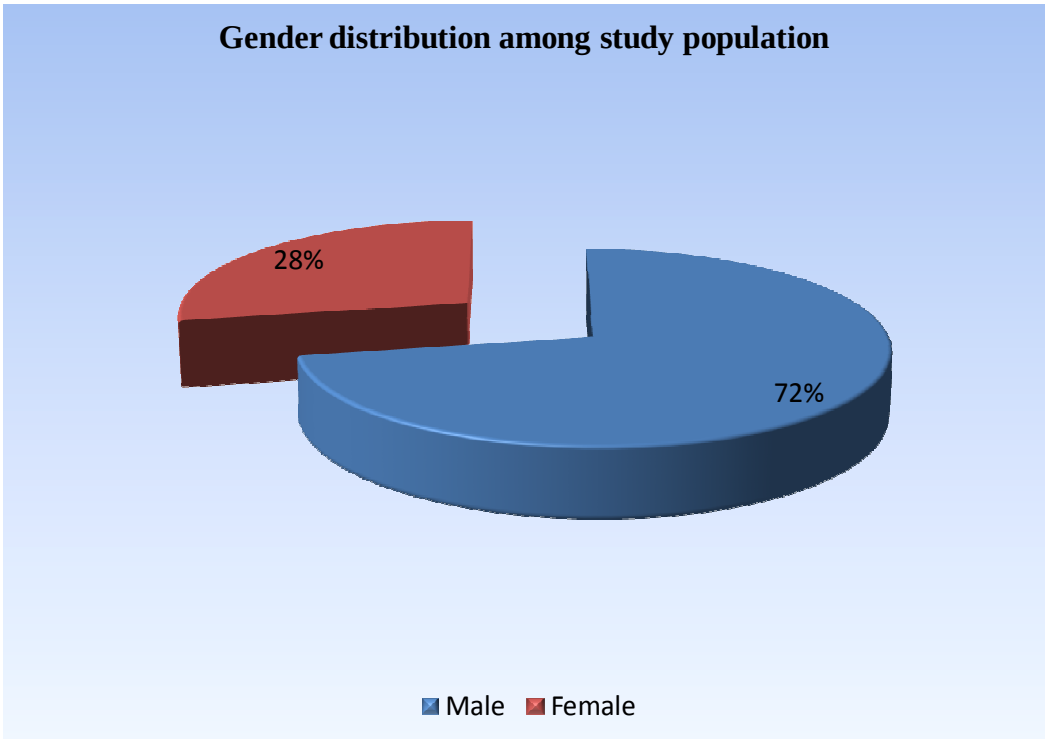


Table3 Distribution of symptom among study population :

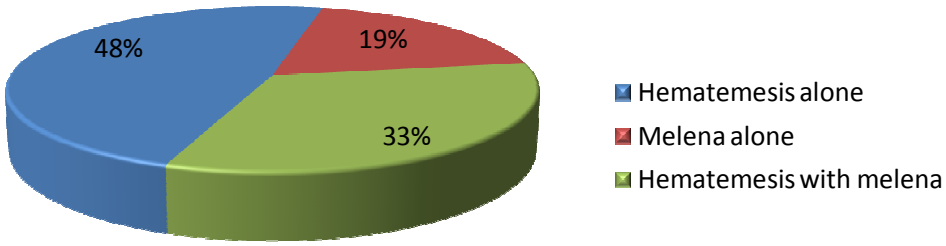
Symptoms	Frequency	Percentage
Hematemesis alone	48	48
Melena alone	19	19
Hematemesis and melena	33	33
Total	100	100

Hematemesis alone occurred in 48% of patients.

Melena alone occurred in 19% of patients.

Both Hematemesis and melena occurred in 33% of patients

Presenting symptoms in study population



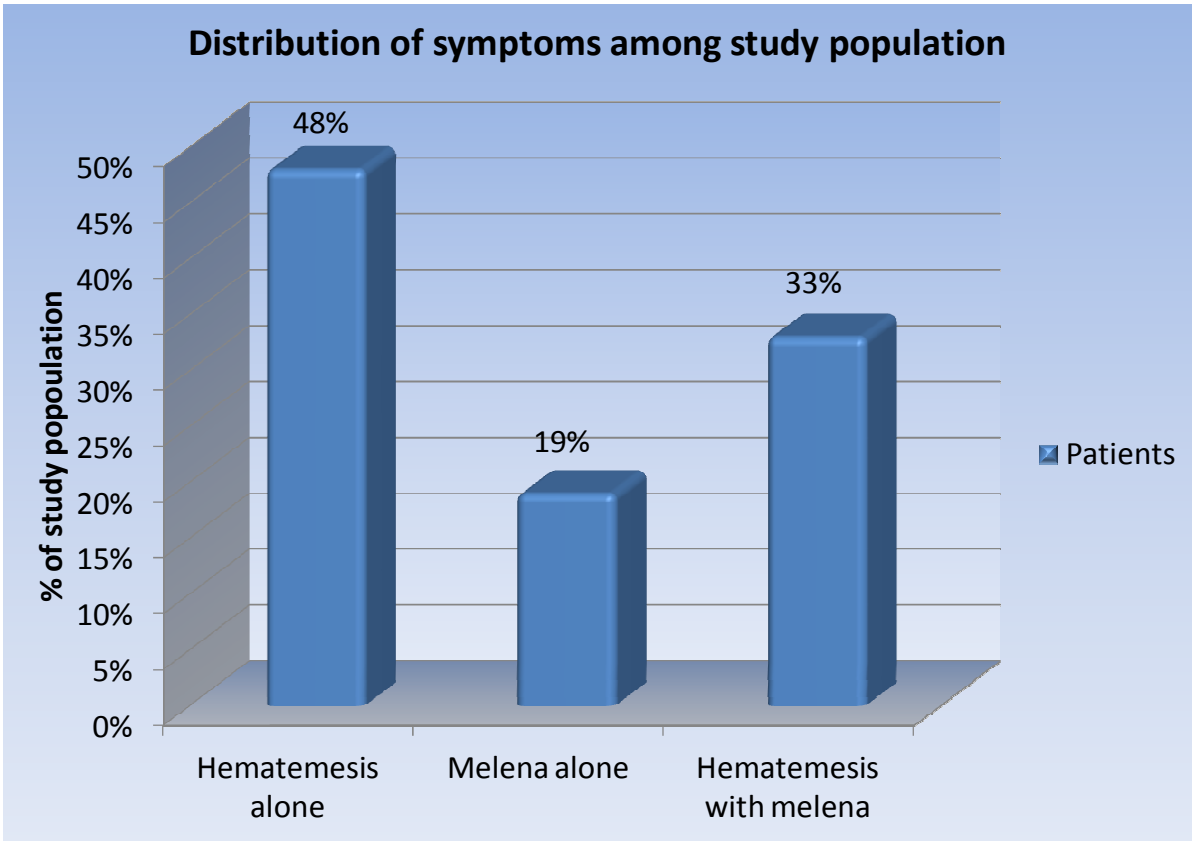
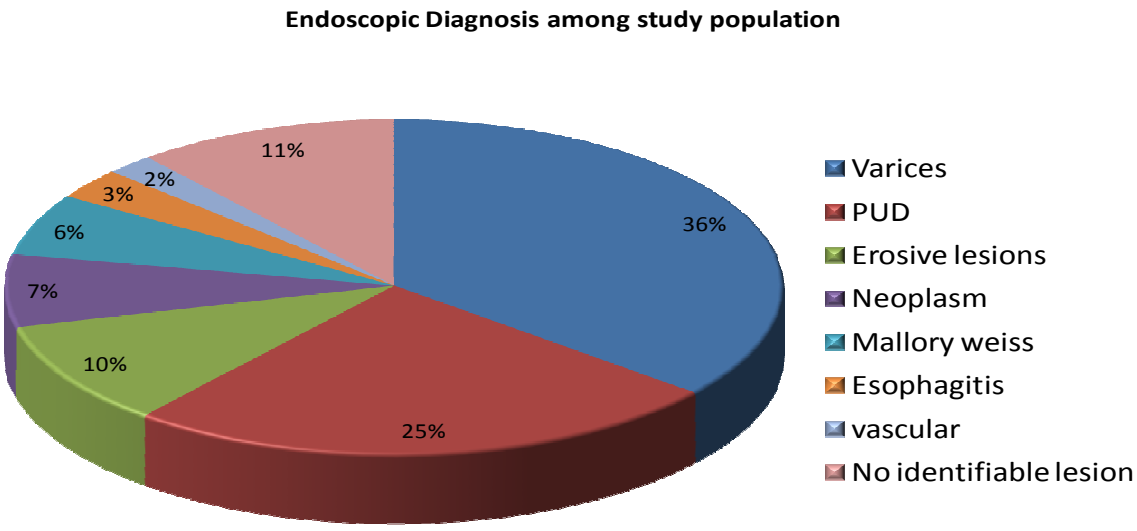

Distribution of symptoms among study population
% of study popoulation
50%
45%
40%
35%
30%
25%
20%
15%
10%
5%
0%
48%
19%
33%
Hematemesis alone
Melena alone
Hematemesis with melena
Patients

Table4 Distribution of Endoscopic diagnosis among study population :

Endoscopic Diagnosis	Frequency	Percentage %
Varices	36	36
PUD	25	25
Erosive gastroduodenal disease	10	10
Esophagitis	3	3
Mallory-Weiss tears	6	6
Neoplasm	7	7
Vascular lesions	2	2
No identifiable lesion	11	11
Total	100	100

Analysing the etiology of upper GI bleed in this study revealed Variceal bleeding as the most common cause in our hospital, which is a tertiary referral center in this region. Variceal bleeding occurred in 36% of patients. Next to varices is the peptic ulcer disease which constituted 25% . Other etiological findings in decreasing order is as Erosive gastroduodenal disease 10%, Neoplasm 7%,Mallory-Weiss tear 6%, Esophagitis 3%, vascular ectasia 2%. No identifiable lesions was found in 11% of the patients enrolled in the study.


Endoscopic Diagnosis among study population
36%
25%
10%
7%
6%
3%
2%
11%
Varices
PUD
Erosive lesions
Neoplasm
Mallory weiss
Esophagitis
vascular
No identifiable lesion

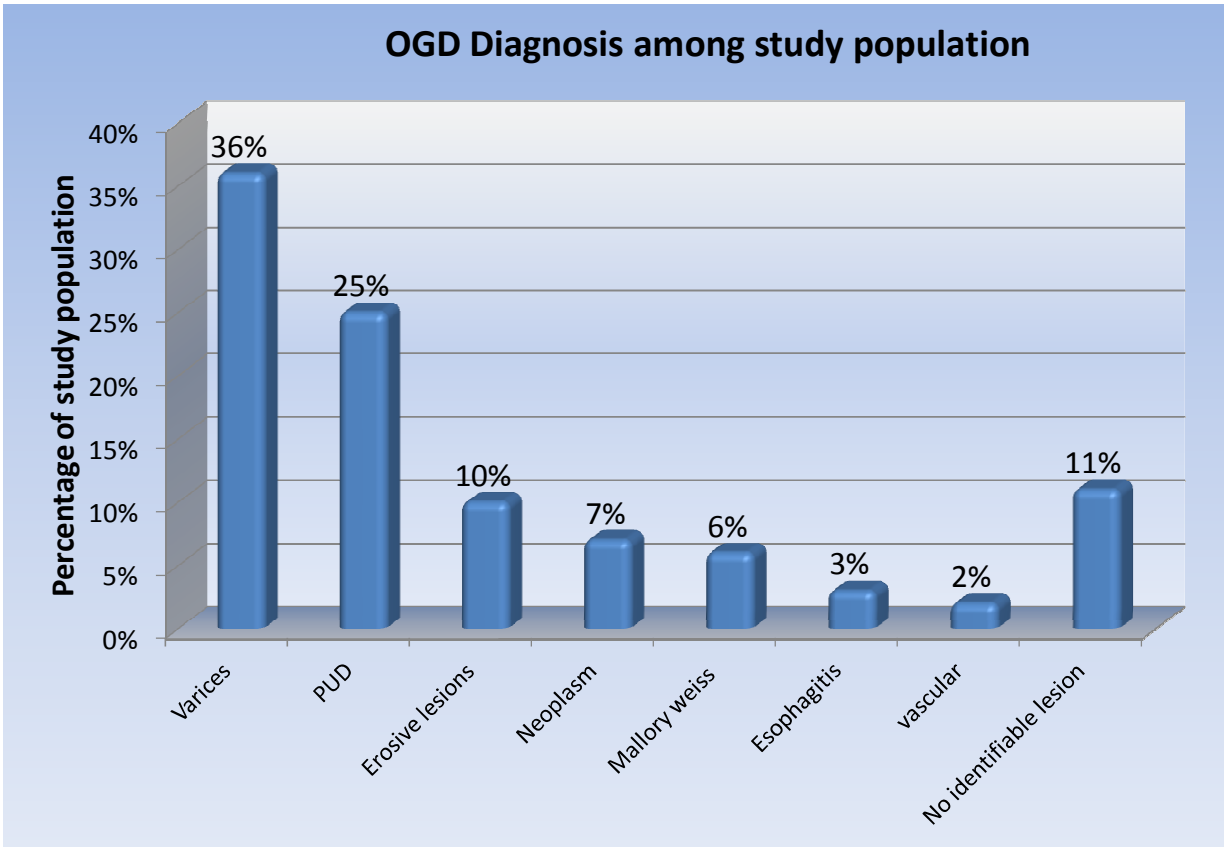
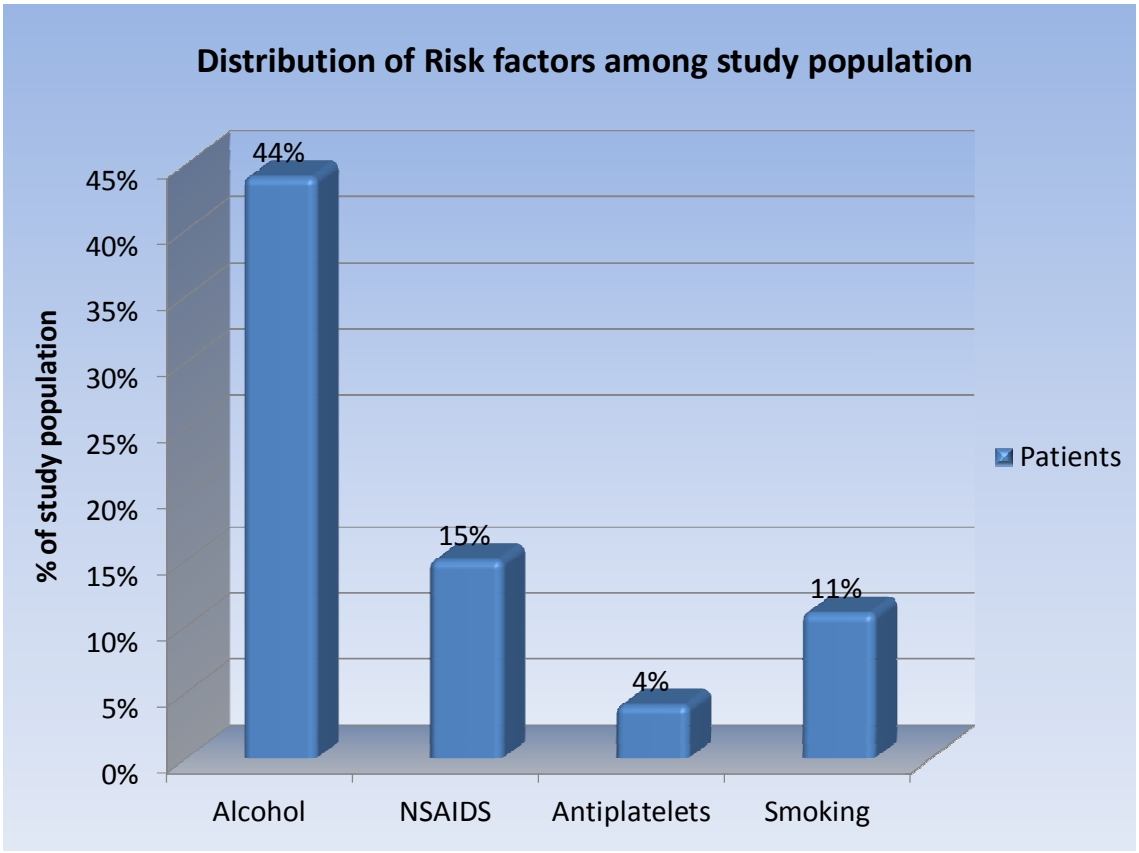

OGD Diagnosis among study population
Percentage of study population
40%
35%
30%
25%
20%
15%
10%
5%
0%
36%
25%
10%
7%
6%
3%
2%
11%
Varices
PUD
Erosive lesions
Neoplasm
Mallory weiss
Esophagitis
vascular
No identifiable lesion

Table5 Distribution of risk factors among study population :

Risk factors	Frequency	Percentage%
Alcohol	44	44%
NSAIDS	15	15%
Antiplatelets	4	4%
Smoking	11	11%



Alcohol :

Alcohol intake was found to be in 44% of patients and absent in 56% of patients. Regarding alcohol and gender relation, alcohol intake was found only among males who were enrolled in this study.

NSAIDs :

NSAIDs intake was found in 15% of patients enrolled in this study.

Antiplatelet :

Antiplatelet drug intake was recorded in 4% of study population.

Smoking :

History of smoking was recorded in 11% of study population and distributed only among male gender.

Table6 Distribution of Co-morbidity among study population :

Co-morbidity	Frequency	Percentage %
CKD	7	7
IHD	9	9
CCF	3	3

Among the study population, co-existing morbidities were found in 19% of patients. Chronic kidney disease was found in 7%, Ischemic heart disease in 9% and Heart failure in 3% of patients.

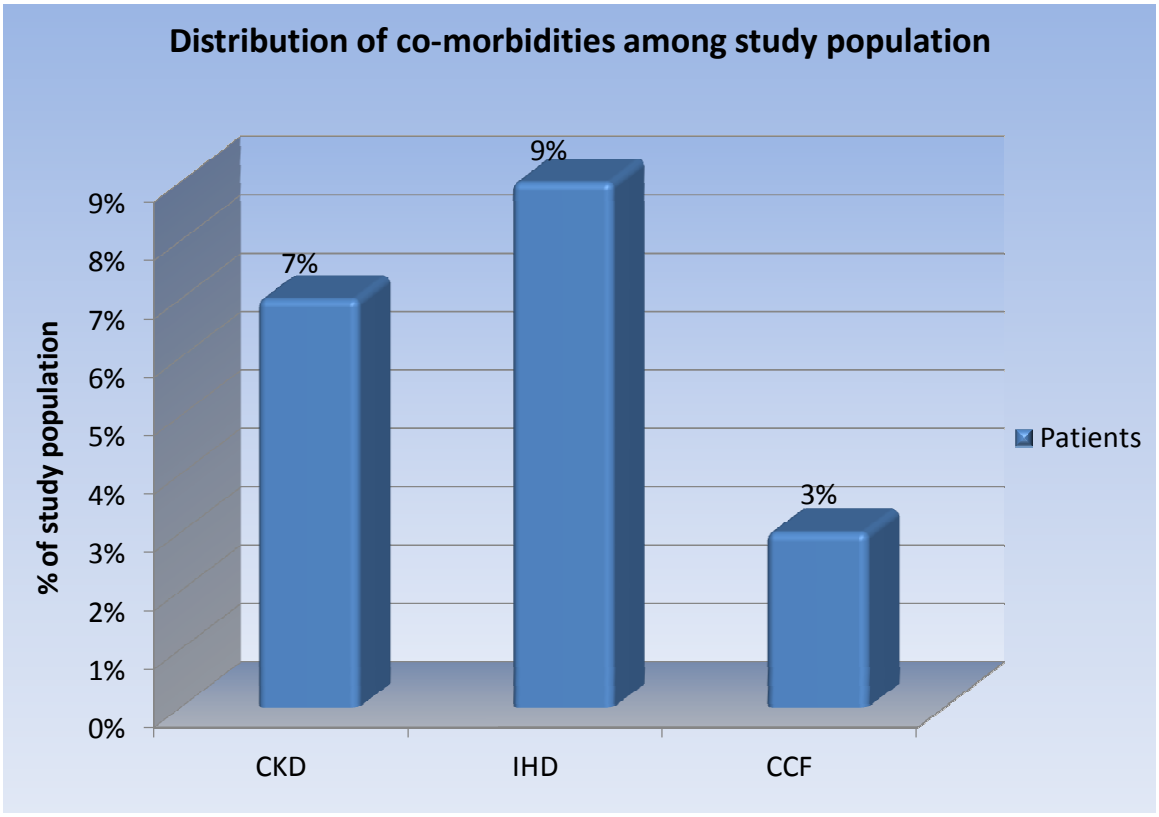
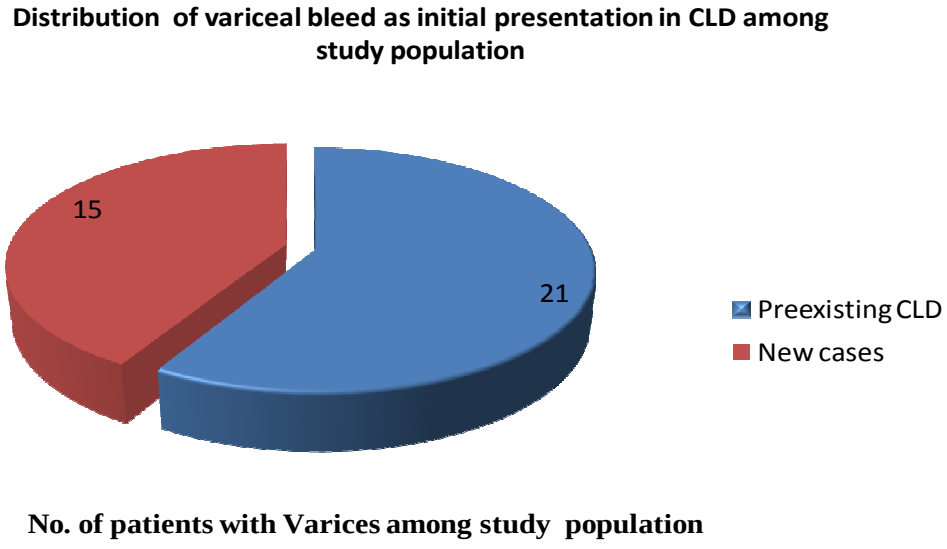


Table7 Preexisting CLD among variceal patients in study population

Preexisting CLD	Frequency	Percentage%
Present	21	21
Absent	79	79
Total	100	100

Information regarding pre-existing liver disease was recorded from history and previous records and found to be in 21% of the study population.

Upper GI bleed as initial presentation in liver disease among study population :



No. of patients with Varices among study population

Table8 Child pugh score among variceal patients in study population

Child pugh score	Frequency	Percentage %
A	15	15
B	16	16
C	5	5
Total	100	100

Severity of liver disease was assessed with Child pugh score in applicable patients. 64 patients of study population are not applicable. Among the rest 36 patients(36%) of patients, Child pugh score A was found in 15 patients(41.6%)),score B in 16(44.4%) and score C in 5(13.8%) .

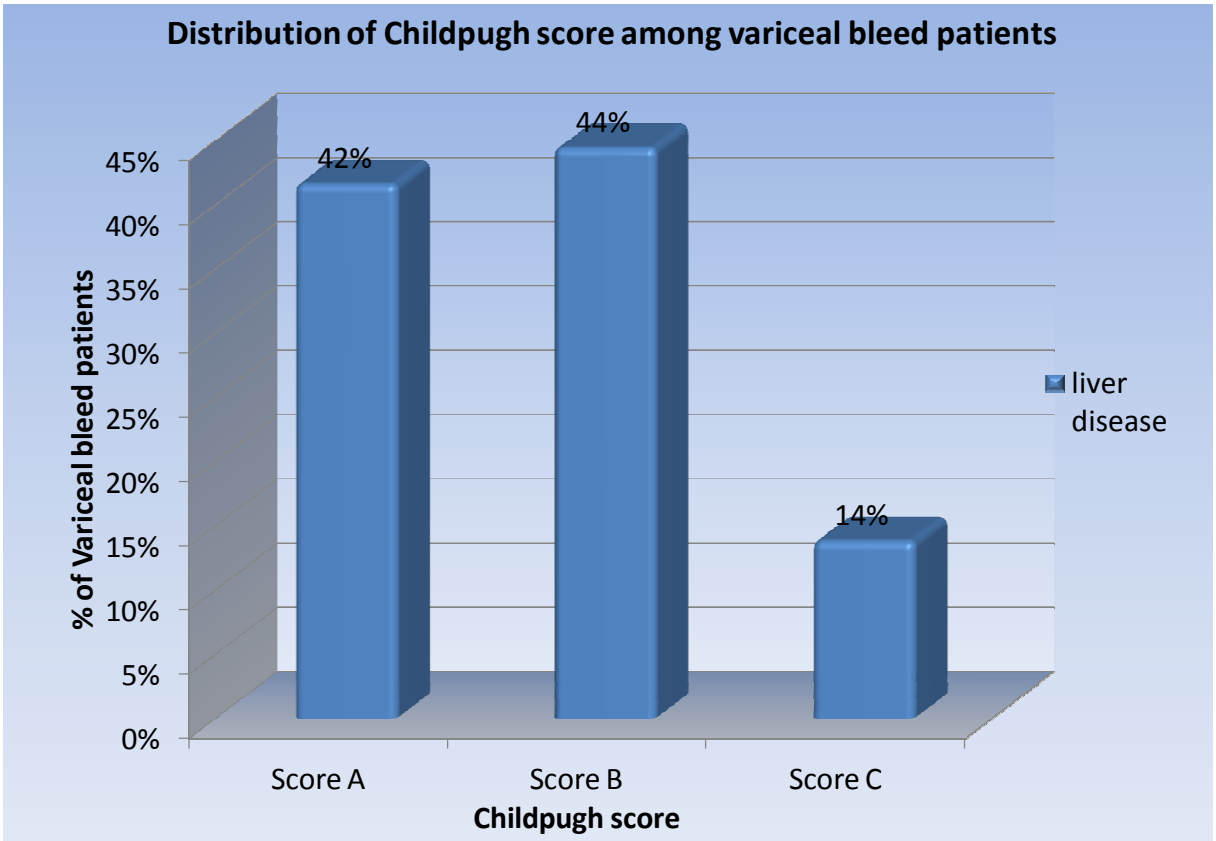

Distribution of Childpugh score among variceal bleed patients
% of Variceal bleed patients
45%
40%
35%
30%
25%
20%
15%
10%
5%
0%
42%
44%
14%
Score A
Score B
Score C
Childpugh score
liver disease

Table9

Relation between Childpugh score and mortality in study population

		Patients	Mortality		P value
		Frequency	Frequency	Percentage %	
Childpugh	A	15	0	0	
	B	16	3	18%	0.007*
	C	5	3	60%	

*Statistically significant

Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	9.810[a]	2	.007
Likelihood Ratio	10.268	2	.006
Linear-by-Linear Association	8.852	1	.003
N of Valid Cases	36		

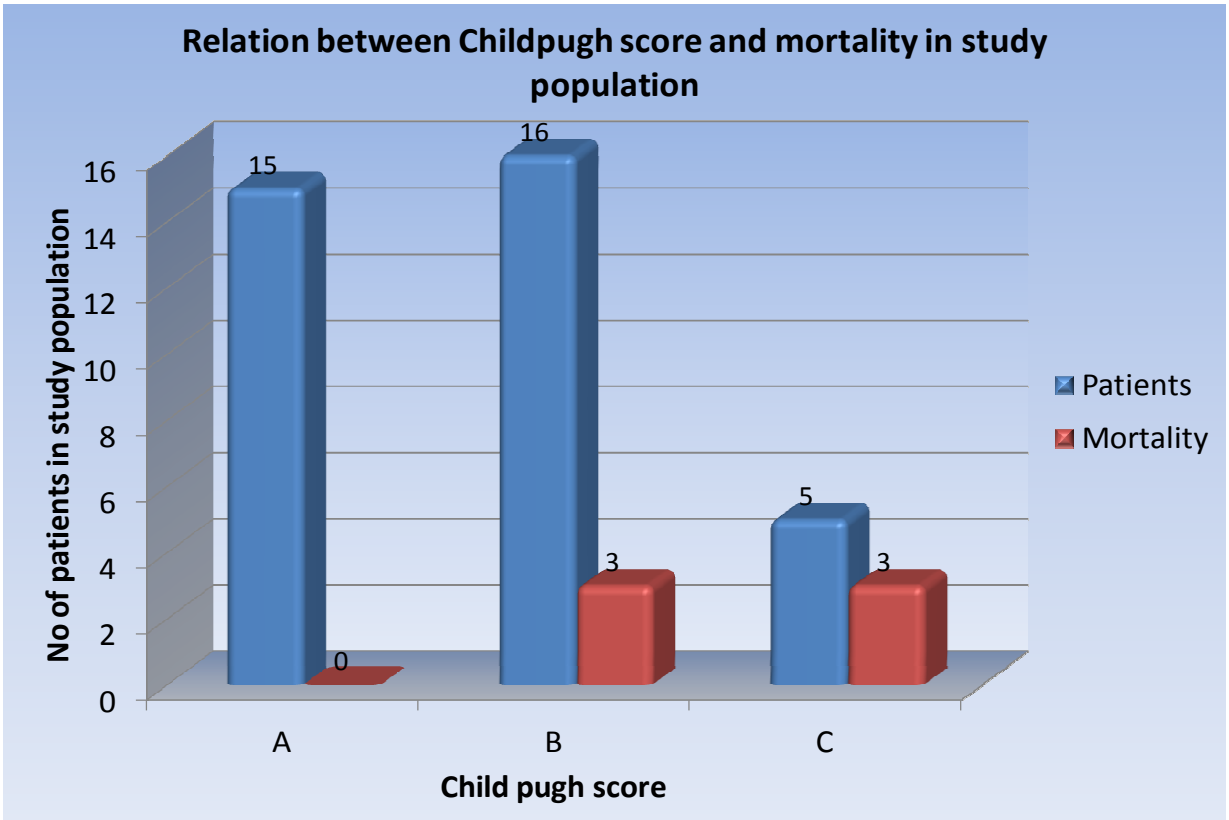

Relation between Childpugh score and mortality in study population
No of patients in study population
16
14
12
10
8
6
4
2
0
15
0
16
3
5
3
A
B
C
Child pugh score
Patients
Mortality

Table10 Distribution of Rockall score in study population:

Rockall score	Frequency	Percentage %
0	20	20
1 – 3	38	38.00%
4 – 7	32	32.00%
>7	10	10.00%
Total	100	100

Risk stratification of patients presenting with upper GI bleed was done with Rockall score. 20% of population falls with score 0, 38% falls within score 1 -3, 32% falls within score 4-7 and 10% falls in the score of greater than 7.

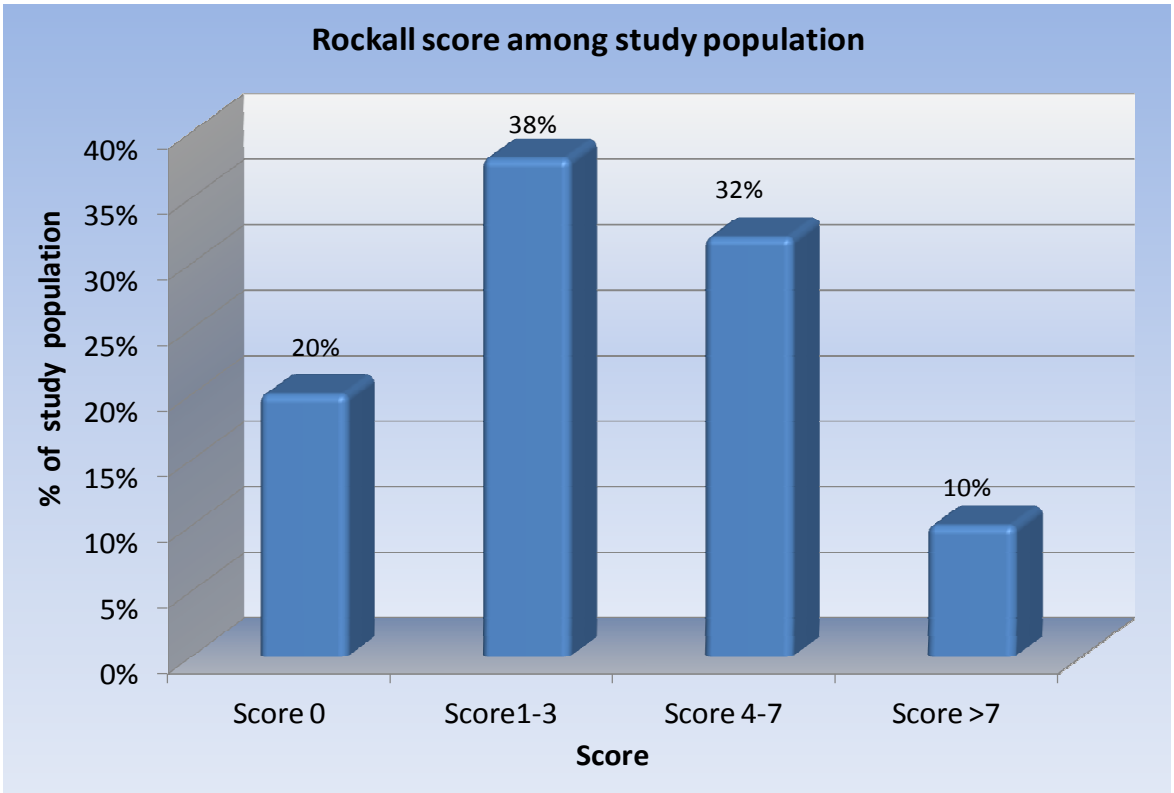


Table11 Relation between Rockall score and rebleed in study population :

		Patients	Rebleed		P value
		Frequency	Frequency	Percentage%	
Rockall score	0	20	0	0	0.001*
	1-3.	38	0	0	
	4-7.	32	10	31.30%	
	>7	10	6	60%	
	Total	100	16	10%	

*Statistically significant

Chi square test	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)	Point probability
Pearson Chi-Square	30.990 a	3	0	0		0.001*
Likelihood Ratio	34.724	3	0	0		
Fisher's Exact Test	29.539			0		
Linear-by-Linear Association	25.671 b	1	0	0	0	
N of Valid Cases	100					

*Statistically significant

Risk of rebleed was assessed with Rockall score. Ther was no bleed among patients with score 0-3. 10 patients(31.3%) had rebleed among 22 with score 4-7. 6 patients(60%) had rebleed among 10 with score >7. Thus rebleed was more with higher score. The association was significant with p value 0.000

Table12 Relation between Rockall score and mortality in study population

		Patients	Mortality		P value
		Frequency	Frequency	Percentage%	
Rockall score	0	20	0	0	0.001*
	1-3.	38	0	0	
	4-7.	32	2	6.30%	
	>7	10	8	80%	

*Statistically significant

Chi square test	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)	Point probability
Pearson Chi-Square	61.389a	3	0	0		
Likelihood Ratio	40.046	3	0	0		
Fisher's Exact Test	34.852			0		
Linear-by-Linear Association	29.470b	1	0	0	0	0
N of Valid Cases	100					

There was no mortality in patients with Rockall score 0 – 3. Among 32 patients with score 4-7, 2 patients expired constituting 6.3% mortality. In patients with score >7, 8 were dead among 10 constituting 80% mortality. Association between rockall score and mortality was significant statistically with P value .001

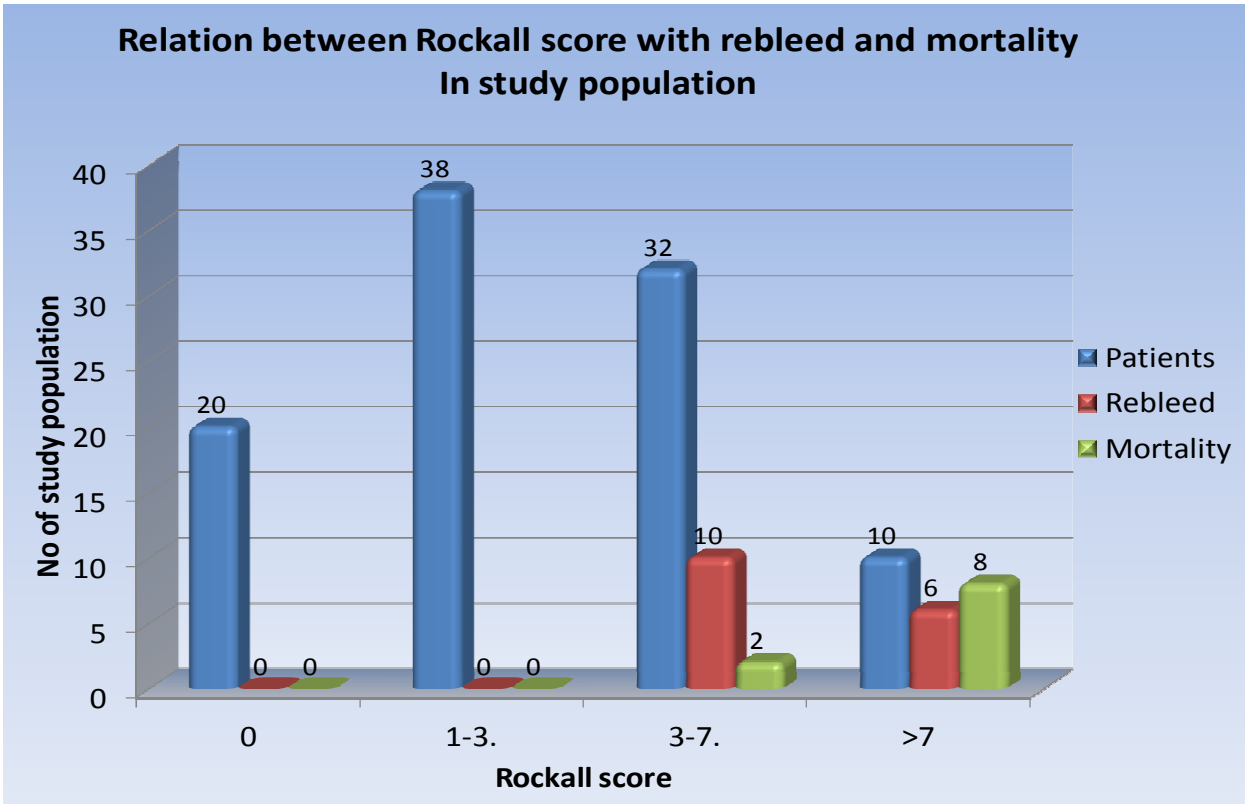

Relation between Rockall score with rebleed and mortality
In study population
No of study population
40
35
30
25
20
15
10
5
0
20
0
0
38
0
0
32
10
2
10
6
8
0
1-3.
3-7.
>7
Rockall score
Patients
Rebleed
Mortality

Table14 Glasgow Blatchford score among study population

GBScore	Frequency	Percentage %
0	26	26
<5	53	53
>5	21	21
Total	100	100

Risk stratification with Glascow Blatchford score shows 26% having score of '0', 53% having score less than 5 and 21% having score greater than 5.

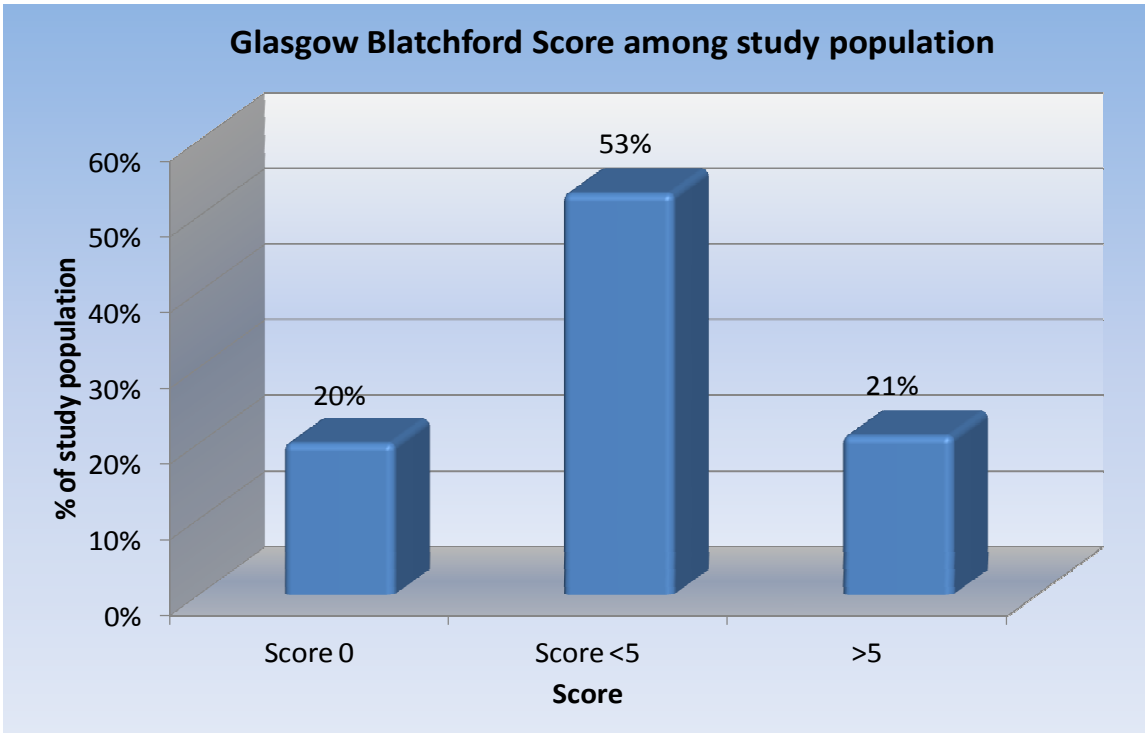


Table15 Relation between Glasgow Blatchford score and rebleed in study population

Chisquare test		Patients	Rebleed		P value
		Frequency	Frequency	Percentage%	
Glasgow Blatchford score	0	26	0	0	0.001*
	<5	53	4	7.50%	
	>5	21	12	57.10%	

*statistically significant

Chi square test	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)	Point probability
Pearson Chi-Square	34.219a	2	0	0		0.001*
Likelihood Ratio	30.89	3	0	0		
Fisher's Exact Test	27.801			0		
Linear-by-Linear Association	25.815b	1	0	0	0	
N of Valid Cases	100					

Patients with score 0 had no rebleed. There was 4 rebleed among 53 patients(7.5%) with score less than 5. 12 rebleed among 21 patients with score 21(57.1%). The association was significant with P value 0.0001

Table16

Relation between Glasgow Blatchford score and mortality in study population:

		Patients	Mortality		P value
		Frequency	Frequency	Percentage %	
Glasgow Blatchford score	0	26	0	0	0.001*
	<5	53	0	0	
	>5	21	10	47.60%	
	Total	100	10	10%	

Chi square test	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)	Point probability
Pearson Chi-Square	41.799a	2	0	0		0.001*
Likelihood Ratio	35.952	2	0	0		
Fisher's Exact Test	30.89			0		
Linear-by-Linear Association	25.941b	1	0	0	0	
N of Valid Cases	100					

Comparing glascow Blatchford score and mortality, there was no death in patients with score 0 and score less than 5. Among 21 patients with score greater than 5, mortality occurred in 10, constituting 47.6% among them. P value was significant .000

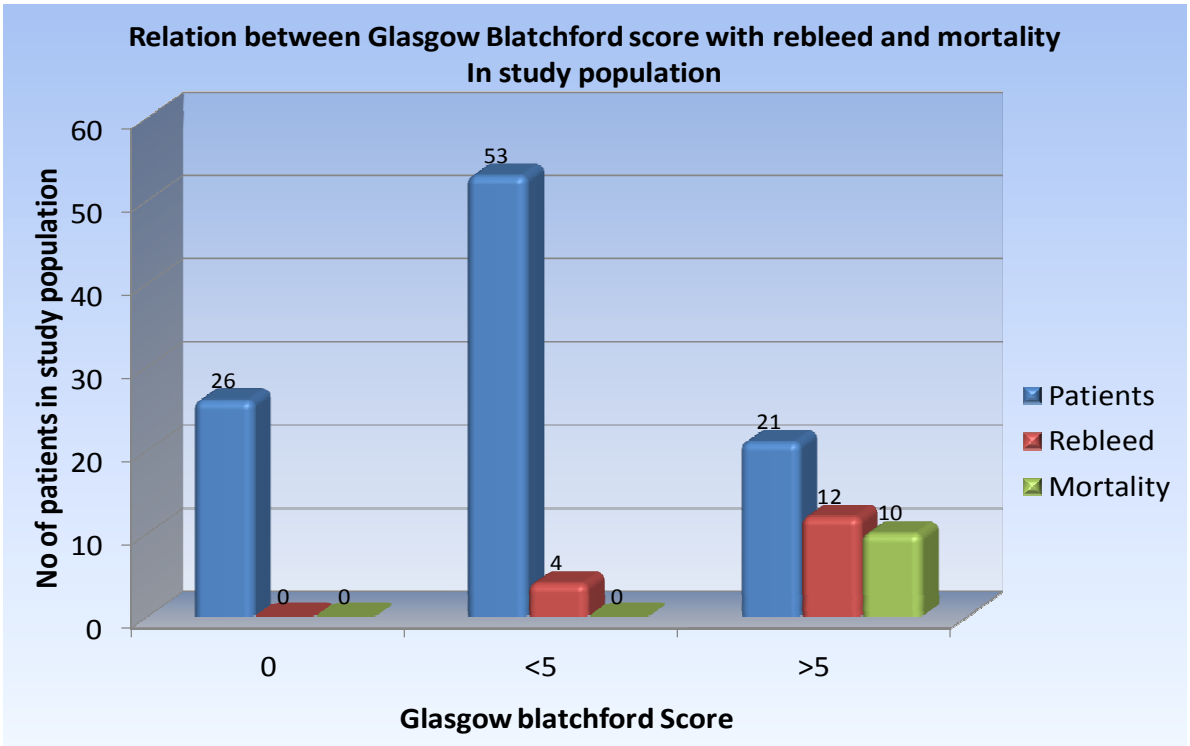

Relation between Glasgow Blatchford score with rebleed and mortality
In study population
No of patients in study population
60
50
40
30
20
10
0
26
0
0
53
4
0
21
12
10
0
<5
>5
Glasgow blatchford Score
Patients
Rebleed
Mortality

Table17 Rebleed among study population:

Rebleed	Frequency	Percentage %
Present	16	16
Absent	84	84

Rebleed after the initial episode and after diagnostic endoscopic procedure was found in 16% patients. 50% of them are due to variceal bleed and other 50% are peptic ulcer related.

Mortality among study population :

Overall all cause mortality occurred in 10% of study population . In this study overall mortality of 9% occurred in males and 1% in females among study population.

Table18 Distribution of mortality in study population

Sex	Patients	Mortality	
	Frequency	Frequency	Percentage%
Male	72	9	12.50%
Female	28	1	3.60%

Among 72 male patients in this study, death occurred in 9 of them constituting 12.5% of death among males. In case of female patients, death occurred in one patient constituting 3.6% of female population.

Table19 Distribution of mortality among various etiology in study population :

Diagnosis	Patients	Mortality	
	Frequency	Frequency	Percentage%
Varices	36	6	16.70%
PUD	25	3	12.00%
Malignacy	7	1	14.30%

In the study population mortality occured in 6 patients (6%)due to variceal bleed, 3 patients(3%) due to PUD and and in one patient (1%) due to neoplasm. Mortality within each etiology shows 16.7% mortality among variceal bleed, 14.3% among neoplasm and 12% among PUD patients.

Table20

Distribution of mortality among co-morbidities in study population :

Diagnosis	Patients	Mortality	
	Frequency	Frequency	Percentage%
CKD	7	4	57.00%
IHD	9	4	44.00%
HF	3	2	66.70%

Among study patients who suffered from CKD, mortality was 57.1%; who had ischemic heart disease mortality was recorded in 44.4% and among heart failure patients death occurred in 66.7%

Discussion

The study conducted with aim of finding the pattern of upper GI bleed in our locality, the results are analysed and showed similarities and variation when compared with similar studies conducted in various parts of India. The results analysed are discussed with relation to each variable below.

Age distribution among study population :

Incidence of UGIB in the study population was more among 40 – 50 years of age, followed by 50 -60 years of age and then 30-40years of age.

In a study done by Rathi *et al*. in Western India the mean age of patients presenting with UGIB was 42 years. In a study by Lakhwani *et al*. in 2000, mean age of patients were 51.9 years

Sex distribution among study population :

Male patients comprised 72% of study population and females 28% of study population.

In Deep Anand *et al* studyUGIB was found to be more common in men (83.33%) as compared to women (16.66%)(2).

Symptoms distribution among study population :

Hematemesis alone was recorded in 48% of patients and melena alone was recorded in 19 % of patients. Both occurred in 33 % of study population.

Deep Anand *et all observed* 27.19% of isolated hematemesis, 64% patients presented with complaints of hematemesis and melena, 12.28% isolated melena, 0.87% patient presented with hematochezia (2).

Etiology of upper GI among study population :

Analying the etiology of upper GI bleed using upper GI scopy revealed varicel bleed as the most common finding constituting 36% of study population. Peptic ulcer disease was the second common cause with 25% of study population. Other findings include Erosive gastroduodenal lesions 10%, Neoplasm 7%, Mallory weiss tear 6%, Esophagitis 3%, other lesions 2%. No identifiable lesion was recorded in 11% of study population.

Referral to tertiary care, patients refusal to evaluation of GI bleed in cases of suspected acute erosive gastritis, higher percentage of alcohol consumption in the study population might contribute to variceal bleed as most common cause.

Study done at Dehradun- Northern india by Deep Anand *et all* revealed 56.14% patients had portal HTN related esophageal and fundal varices, 14.91% had gastric and duodenal ulcer, 12.28% had gastric erosions/gastritis, 8.77% had Mallory–Weis tear, 4.38% had gastric malignancy(2)

Anand *et al*. from North India, causes of bleeding were esophageal varices in 45.5%, duodenal ulcer in 25%, gastric ulcer in 5% and gastritis in 8.5%.[12

Dilawari *et al*. found variceal bleeding due to portal hypertension (36%) as the most frequent cause followed by peptic ulceration (24%) and gastric erosions (19%).[13

Differing with other studies, was the one done at coastal odisha in 2013, most common cause in endoscopic diagnosis was duodenal ulcer in 57.6% patients, variceal bleed in 12.8%, gastric ulcer in 1.8% , Mallory–Weiss tear in 1.8% , erosive gastritis in 1.8% patients and malignancy comprised of 7.7%[8

Risk factors :

Alcohol was found as risk factor among the population. 44% study population had alcohol. 15% had history of NSAIDs intake. 4% had Antiplatelet drug. History of Smoking was recorded in 11% .

Comorbidities :

The morbidity and mortality was more when associated co morbid conditions existed. CKD was recorded in 7%, Ischemic heart disease in 9%, Heart failure in 3% of study population.

Chronic liver disease :

Pre-existing liver disease was recorded in 21%(21 patients) of study population. Among 36 patient with variceal bleed, 14(38.8%)were new patients who had not been diagnosed to have liver disease previously. The initial presenting feature which made them sought medical advice is the UGIB.

Child pugh score :

Severity of liver disease was assessed with Child pugh score in patients with variceal bleed. Child pugh score A was found in 15%,score B in 16% and score C in 5% .

ROCKALL SCORE :

Risk stratification of patients presenting with upper GI bleed was done with Rockall score. Risk of rebleed and mortality was found to be increased in patients with score 4-7 and much higher in score >7.

Ther was no re bleed and mortality among patients with score 0-3.The association was significant with p value 0.001

In various validating studies done around the world, Rockall score of 0-3 has no intervention needed, score 4-7 had good outcome with minimal intervention and score more than 7 had significant hospital stay, rebleed and mortality.

Glasgow Blatchford Score :

Risk stratification with Glasgow Blatchford score shows that patient with score 0 had no rebleed, no mortality and required no intervention. There was no mortality in patients with score less than 5 and less intervention was needed. Patients with score >5 had significant rebleed and mortality which was statistically significant.

In the original study done by Blatchford, the intervention and hospital stay was significantly low in score group 0.

Mortality :

Overall mortality occurred in 10% of patients. 6% occurred due to variceal bleed, 3% due to peptic ulcer disease and 1% due to neoplasm. Mortality within each category revealed higher percent among variceal bleed followed by neoplasm.

Mortality was associated more with patients who suffered other organ failure.

Anand *et all* study shows overall mortality of 21% among study population, with portal hypertensive group being the common followed by peptic ulcer disease.

In a study by Chalasani *et al*.[22] a total of 231 subjects were included, and their in‐hospital, 6‐week, and overall mortality rates were 14.2%, 17.5%, and 33.5%, respectively

Limitations of the study :

This study has its own limitations. The study was conducted with minimal sample population. The time of presentation varied among patients and subsequently the timing of endoscopy evaluation and intervention. Outcome of the study is influenced by patients who did not consent or did not undergo upper GI scopy.

Summary

This study done in our hospital,a tertiary care center to southern part of the state, revealed that the most common cause of Upper Gastrointestinal bleeding was Varices. The second most common cause was Peptic ulcer related bleed. The other causes in descending order of frequency were Erosive gastroduodenal lesions, Neoplasm, Mallory-Weiss tear, Esophaghitis, Vascular lesions.

Alcohol being the associated risk factor in variceal bleed, accounted for common cause in portal hypertension. This reflects the burden of alcohol consumption on health issues.

This study revealed sex ratio of male to female 2.5:1. Male patients in the study was comprised of 72%

This study shows the majority of patients(29%) fall in the age group of 40-50 years, followed by 50-60years of age(25%).

Among the study population, the patients who had melena had statistically significantly asscociation with mortality. Mortality occurred in patients who had hematemesis alone, but significant association could not be established.

In this study,risk of rebleed and mortality was significantly absent in patients with ROCKALL score of 0-3 and Glasgow Blatchford score of 0 .

The risk was significantly increased with higher scores in both the system.

Among the study population, factors associated with mortality were presence of co-morbidities like renal failure and cardiac failure. Mortality was associated with Rockall score of >7 and Glasgow Blatchford score of >5 which were statistically significant.

Conclusion

1. Variceal bleed is the most common cause in this study reflecting upon the population of this region.

2. Small percent of liver disease manifested with upper gastrointestinal bleed as initial presentation.

3.Patient risk stratification for intervention and can be prioritised with scoring system. Prognosis is poor in patients with co-morbidities and with higher score in scoring system.

Annexures

Bibilography :

1. Wilkins T, Khan N, Nabh A, Schade RR. Diagnosis and management of upper gastrointestinal bleeding. Am Fam Physician. 2012 Mar;85(5):469–76.
2. Gupta R, Ahuja V, Anand D, Dhar M. Clinical and endoscopic profile of patients with upper gastrointestinal bleeding at tertiary care center of North India. J Dig Endosc [Internet]. 2014;5(4):139. Available from: http://www.jdeonline.in/text.asp?2014/5/4/139/150660
3. Odisha C. Original Article. 2013;34(1):14–7.
4. Harrison’s Principles of Internal Medicine, 19E (2015) [True PDF] [UnitedVRG].
5. Al-Busafi S a, McNabb-Baltar J, Farag A, Hilzenrat N. Clinical Manifestations of Portal Hypertension. Int J Hepatol [Internet]. 2012;2012:1–10. Available from: http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=3457672&tool=pmcentrez&rendertype=abstract
6. Cholongitas E, Papatheodoridis G V, Vangeli M, Terreni N, Patch D, Burroughs AK. Systematic review: The model for end-stage liver disease--should it replace Child-Pugh’s classification for assessing prognosis in cirrhosis? Aliment Pharmacol Ther. 2005 Dec;22(11–12):1079–89.
7. Blair SD, Janvrin SB, McCollum CN, Greenhalgh RM. Effect of early blood transfusion on gastrointestinal haemorrhage. Br J Surg. 1986 Oct;73(10):783–5.
8. Fundamentals T. Practical Gastrointestinal Endoscopy.
9. Laine L, Jensen DM. Management of patients with ulcer bleeding. Am J Gastroenterol [Internet]. 2012;107(3):345–60; quiz 361. Available from: http://www.ncbi.nlm.nih.gov/pubmed/22310222
10. Rockall TA, Logan RF, Devlin HB, Northfield TC. Risk assessment after acute upper gastrointestinal haemorrhage. Gut [Internet]. 1996;38(3):316–21. Available from: http://www.ncbi.nlm.nih.gov/pubmed/8675081\nhttp://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=PMC1383057
11. Blatchford O, Murray WR, Blatchford M. A risk score to predict need for

treatment for upper-gastrointestinal haemorrhage. Lancet (London, England) [Internet]. 2000;356(9238):1318–21. Available from: http://www.ncbi.nlm.nih.gov/pubmed/11073021

12. Paquet KJ. Endoscopic paravariceal injection sclerotherapy of the esophagus--indications, technique, complications: results of a period of 14 years. Gastrointest Endosc. 1983 Nov;29(4):310–5.
13. AYRES SJ, GOFF JS, WARREN GH. Endoscopic Sclerotherapy for Bleeding Esophageal Varices: Effects and Complications. Ann Intern Med [Internet]. 1983 Jun 1;98(6):900–3. Available from: http://dx.doi.org/10.7326/0003-4819-98-6-900
14. Stiegmann G V, Goff JS, Michaletz-Onody PA, Korula J, Lieberman D, Saeed ZA, et al. Endoscopic sclerotherapy as compared with endoscopic ligation for bleeding esophageal varices. N Engl J Med. 1992 Jun;326(23):1527–32.
15. Gimson AE, Ramage JK, Panos MZ, Hayllar K, Harrison PM, Williams R, et al. Randomised trial of variceal banding ligation versus injection sclerotherapy for bleeding oesophageal varices. Lancet (London, England). 1993 Aug;342(8868):391–4.
16. Binmoeller KF, Soehendra N. “Superglue”: the answer to variceal bleeding and fundal varices? Vol. 27, Endoscopy. GERMANY; 1995. p. 392–6.
17. Stanley AJ, Ashley D, Dalton HR, Mowat C, Gaya DR, Thompson E, Warshow U, Groome M, Cahill A, Benson G, et al. Outpatient management of patients with low-risk upper-gastrointestinal haemorrhage: multicentre validation and prospective evaluation. Lancet. 2009;373:42–47. [PubMed]

18. Chan JCH, Ayaru L. Analysis of risk scoring for the outpatient management of acute upper GI bleeding. Frontline Gastroenterol. 2011;2:19–25.

19. Chen IC, Hung MS, Chiu TF, Chen JC, Hsiao CT. Risk scoring systems to predict need for clinical intervention for patients with nonvariceal upper gastrointestinal tract bleeding. Am J Emerg Med. 2007;25:774–779. [PubMed]

20. Le Jeune IR, Gordon AL, Farrugia D, Manwani R, Guha IN, James MW. Safe discharge of patients with low-risk upper gastrointestinal bleeding (UGIB): can the use of Glasgow-Blatchford Bleeding Score be extended? Acute Med. 2011;10:176–181. [PubMed]

21. Pang SH, Ching JY, Lau JY, Sung JJ, Graham DY, Chan FK. Comparing the Blatchford and pre-endoscopic Rockall score in predicting the need for endoscopic therapy in patients with upper GI hemorrhage. Gastrointest Endosc. 2010;71:1134–1140. [PubMed]

22. Rockall TA, Logan RF, Devlin HB, Northfield TC. Selection of patients for early discharge or outpatient care after acute upper gastrointestinal haemorrhage. National Audit of Acute Upper Gastrointestinal Haemorrhage. Lancet. 1996;347:1138–1140. [PubMed]

23. Blair SD, Janvrin SB, McCollum CN, et al. Effect of early blood transfusion on gastrointestinal

haemorrhage. Br J Surg 1986;73(10):783–5.

24.R. J. Groszmann, G. Garcia-Tsao, J. Bosch et al., "Beta-blockersto prevent gastroesophageal varices in patients with cirrhosis,"

New England Journal of Medicine, vol. 353, no. 21, pp. 2254–2261, 2005.

25. M. Merli, G. Nicolini, S. Angeloni et al., "Incidence and natural history of small esophageal varices in cirrhotic patients,"*Journal of Hepatology*, vol. 38, no. 3, pp. 266–272, 2003.

26. L. T. Sumanovski, E. Battegay, M. Stumm, M. Van Der Kooij,and C. C. Sieber, "Increased angiogenesis in portal hypertensiverats: role of nitric oxide," *Hepatology*, vol. 29, no. 4, pp.

1044–1049, 1999.

27. A. Vianna, P. C. Hayes, G. Moscoso et al., "Normal venouscirculation of the gastroesophageal junction: a route to understandingvarices," *Gastroenterology*, vol. 93, no. 4, pp. 876–889,1987.

28. G. Garcia-Tsao, R. J. Groszmann, and R. L. Fisher, "Portalpressure, presence of gastroesophageal varices and varicealbleeding," *Hepatology*, vol. 5, no. 3, pp. 419–424, 1985.

29. R. de Franchis, J. P. Pascal, E. Ancona et al., "Definitions,methodology and therapeutic strategies in portal hypertension.A Consensus Development Workshop, Baveno, LakeMaggiore, Italy, April 5 and 6, 1990," *Journal of Hepatology*,vol. 15, no. 1-2, pp. 256–261, 1992.

30. E. Brocchi, G. Caletti, G. Brambilla et al., "Prediction of the first variceal hemorrhage in patients with cirrhosis of the liver and esophageal varices. A prospective multicenter study," *NewEngland Journal of Medicine*, vol. 319, no. 15, pp. 983–989,1988.

31. F. Nevens, R. Bustami, I. Scheys, E. Lesaffre, and J. Fevery,“Variceal pressure is a factor predicting the risk of a first variceal bleeding: a prospective cohort study in cirrhotic patients,” *Hepatology*, vol. 27, no. 1, pp. 15–19, 1998.

32. G. D’Amico and R. De Franchis, “Upper digestive bleeding in cirrhosis. Post-therapeutic outcome and prognostic indicators,”*Hepatology*, vol. 38, no. 3, pp. 599–612, 2003.

33. S. K. Sarin, D. Lahoti, S. P. Saxena, N. S. Murthy, and U. K Makwana, “Prevalence, classification and natural history of gastric varices: a long- term follow-up study in 568 portal hypertension patients,” *Hepatology*, vol. 16, no. 6, pp. 1343–1349, 1992.

34. T. Kim, H. Shijo, H. Kokawa et al., “Risk factors for hemorrhage from gastric fundal varices,” *Hepatology*, vol. 25, no. 2,pp. 307–312, 1997.

35. D. Lebrec and J. P. Benhamou, “Ectopic varices in portal hypertension,” *Clinics in Gastroenterology*, vol. 14, no. 1, pp.105–121, 1985.

36. M. Kinkhabwala, A. Mousavi, S. Iyer, and R. Adamsons,“Bleeding ileal varicosity demonstrated by transhepatic portography,”*American Journal of Roentgenology*, vol. 129, no. 3,pp. 514–516, 1977.

37. F. Khouqeer, C.Morrow, and P. Jordan, “Duodenal varices as acause ofmassive upper gastrointestinal bleeding,” *Surgery*, vol.102, no. 3, pp. 548–552, 1987.

38. N.Watanabe, A. Toyonaga, S. Kojima et al., “Current status ofectopic varices in Japan: results of a survey by the Japan Societyfor Portal Hypertension,” *Hepatology Research*, vol. 40, no. 8,pp. 763–776, 2010.

39. M. Farid and M. A. ElHoda, “Anorectal varices endoscopic dilemma. Joint Euro-Asian Congress of Endoscopic Surgery,”*Surgical Endoscopy*, pp. 445–877, 1997.

40. M. A. Almadi, A. Almessabi, P. Wong, P. M. Ghali, and A.Barkun, “Ectopic varices,” *Gastrointestinal Endoscopy*, vol. 74,no. 2, pp. 380–388, 2011.

PROFORMA

Name :

Age / sex : IP.No :

Residence : Ward :

Occupation :

Socioeconomic status :

Complaints of : duration :

Present complaints:

H/o haemetemesis

duration

no. of episodes

amount of blood in vomitus

H/o melena

duration

H/o epigastric pain

H/o fever

H/o other bleeding manifestations

H/o loss of appetite / weight

Personal h/o :

H/o alcohol intake

years amount/week last intake

H/o smoking

Past h/o :

H/o NSAIDS / drug intake

H/o DM

H/o Hypertension

H/o coexisting diseases

General examination:

Vitals :

Pulse :

BP :

Systemic examination:

ABDOMEN

CVS

RS

CNS

Investigations:

Hb

TC DC

Platelet count

RBS

LFT

SGOT SGPT ALP S.Bilirubin S.Proteins

PT INR

RFT

ULTRASOUND ABDOMEN :

UPPER GI SCOPY :

Rockall score :

Glasgow Blatchford score :

Child pugh score :

Abbrevations :

CLD – Chronic liver disease

EST – Endoscopic sclerotherapy

EVL – Endoscopic variceal ligation

GI – Gastrointestinal

GBS – Glasgow Blatchford score

NSAIDs – Non steroidal anti inflammatory drugs

OGD – oesophago gastro duodenoscopy

PPI – Proton pump inhibitors

PUD – Peptic ulcer disease

Master chart :

Name	Age	sex	Hematemesis	Melena	OGD finding	Alcohol	NSAIDs	Anti platelet	Smoking	Co morbidity	Pre existing CLD	Child pugh score	Rockall score	GBS	Rebleed	Mortality
Mayandi	5	1	1	0	1	0	0	1	0	2	1	1	2	2	1	0
Mariappan	4	1	1	1	2	0	0	0	0	0	0	0	2	2	1	0
Nallamal	5	2	0	1	4	0	1	0	0	1	0	0	1	2	0	0
Balusamy	5	1	0	1	2	0	1	0	0	0	0	0	0	2	0	0
Govindhan	5	1	1	1	1	1	0	0	0	0	1	1	2	3	1	0
Karuppu	4	1	1	0	1	1	0	0	0	0	1	2	1	2	0	0
Bachiyam	3	2	1	1	2	0	0	0	0	0	0	0	1	2	0	0
Deepa	1	2	1	0	8	1	0	0	0	0	0	0	0	1	0	0
Murugan	3	1	1	0	6	1	0	0	1	0	0	0	1	2	0	0
Kalyani	4	1	0	1	1	1	0	0	0	0	0	1	1	2	0	0
Harimurugan	4	1	1	0	1	1	0	0	0	0	0	1	1	2	0	0
Panjan	5	1	1	0	2	1	0	0	1	0	0	0	0	1	0	0
Raja	2	1	1	1	5	1	0	0	0	0	0	0	0	1	0	0
Alagarsamy	7	1	1	1	2	0	1	0	0	1	0	0	3	3	1	1
Kumar	3	1	1	0	4	1	0	0	0	0	0	0	0	2	0	0
Thiru	4	1	1	0	5	1	0	0	0	0	0	0	0	2	0	0
Kanagaraj	6	1	1	1	1	1	0	0	0	3	1	2	3	3	1	1
Rakammal	4	2	1	0	2	0	1	0	0	0	0	0	1	2	0	0
Saleem	4	1	1	0	3	0	0	0	1	0	0	0	1	1	0	0
Pitchai	6	1	1	1	6	1	0	0	1	2	0	0	3	3	0	1
Indurani	6	2	1	0	1	0	0	0	0	0	0	2	2	2	0	0
Karthik	3	1	0	1	3	0	0	0	0	0	0	0	1	1	0	0
Irulappan	6	1	0	1	2	0	0	1	0	2	0	0	2	2	0	0
Kandappan	4	1	1	0	2	0	0	0	0	0	0	0	0	1	0	0
yasodhai	5	2	0	1	1	0	0	0	0	0	0	1	2	2	0	0
Chinnakutty	6	1	1	1	1	0	0	0	0	2	1	3	3	3	1	1
Rajendran	5	1	1	0	3	0	0	1	0	0	0	0	1	2	0	0
Arumugam	3	1	1	0	7	0	0	0	0	0	0	0	0	1	0	0
Lilysiromani	5	2	1	1	7	0	0	0	0	0	0	0	2	2	0	0
Pappathi	2	2	1	0	5	0	0	0	0	1	0	0	1	2	0	0
Prabanjan	2	1	1	0	8	0	0	0	0	0	0	0	0	1	0	0
Lakshmi	7	2	1	1	2	0	0	0	0	1	0	0	3	3	1	1
Amutha	3	2	1	1	1	1	0	0	0	0	0	1	1	2	0	0
Indra	2	2	1	0	8	0	0	0	0	0	0	0	0	1	0	0
Palanisamy	5	1	1	1	6	1	0	0	1	0	0	0	2	2	0	0
Arumugam	4	1	1	1	1	1	0	0	0	0	1	2	2	2	0	0
yogapriya	1	2	1	0	8	0	0	0	0	0	0	0	0	1	0	0
Arumugam	4	1	0	1	1	1	0	0	0	2	1	2	2	2	0	0
Rangan	4	1	1	0	1	0	0	0	0	0	0	1	1	2	0	0
kumar	3	1	1	0	8	0	0	0	0	0	0	0	1	1	0	0
sumathi	2	2	1	0	8	0	0	0	0	0	0	0	0	1	0	0
Muthusamy	5	1	0	1	2	0	1	0	0	1	0	0	2	3	1	1
Karuppaiya	4	1	1	1	1	1	0	0	0	0	0	1	1	2	0	0
Narayanan	5	1	1	1	1	1	0	0	1	2	1	2	3	3	0	1
Rajesh	2	1	1	1	2	1	0	0	0	0	0	0	1	1	0	0
Muthu	2	1	1	0	3	1	0	0	0	0	0	0	1	1	0	0
Alagarsamy	5	1	1	0	6	1	0	0	1	0	0	0	2	2	0	0
karuppu	5	1	0	1	1	1	0	0	1	3	1	2	3	3	1	1

Mohan	4	1	1	0	1	1	0	0	0	0	0	2	2	2	0	0
Rajesh	5	1	0	1	1	1	0	0	0	0	1	3	2	3	0	0
Naveen	3	1	1	1	5	1	0	0	0	0	0	0	1	2	0	0
Samikannu	4	1	1	1	2	0	1	0	0	1	0	0	3	3	1	0
Paulsamy	3	1	1	0	4	1	0	0	0	0	0	0	0	1	0	0
Gopinathan	3	1	1	0	5	1	0	0	0	0	0	0	1	2	0	0
Savarimuithu	5	1	0	1	1	1	0	0	0	0	1	3	2	3	1	0
Sathish	4	1	1	0	2	1	0	0	0	0	0	0	0	1	0	0
Pitchaikani	5	1	1	0	1	0	0	0	0	0	1	2	2	3	0	0
Hanifa	5	1	1	1	6	1	0	0	1	2	0	0	2	3	0	0
Balamurugan	4	1	1	0	1	0	0	0	0	0	0	1	2	2	0	0
Chinthamani	3	2	0	1	3	0	1	0	0	0	0	0	1	2	0	0
Sandhiya	3	2	0	1	2	0	0	0	0	0	0	0	1	2	0	0
Selvi	2	2	1	0	8	0	0	0	0	0	0	0	0	1	0	0
Ganga	3	2	0	1	3	0	1	0	0	0	0	0	1	2	0	0
Pandi	4	1	1	1	1	0	0	0	0	0	0	1	2	2	0	0
Subbaiya	5	1	1	1	2	0	1	0	0	0	0	0	2	2	1	0
Moorthy	3	1	1	0	5	0	0	0	0	0	0	0	0	1	0	0
Dinesh	2	1	1	1	3	0	0	0	1	0	0	0	1	1	0	0
Muthupandi	4	1	1	0	1	1	0	0	0	0	1	2	3	3	0	0
Selvam	3	1	1	0	8	0	0	0	0	0	0	0	0	1	0	0
Rangasamy	4	1	1	1	2	0	0	0	0	0	0	0	1	2	0	0
Meena	3	2	1	1	1	0	0	0	0	0	1	2	2	3	0	0
Aruna	3	2	1	0	3	0	1	0	0	0	0	0	1	1	0	0
Rani	4	2	1	1	6	0	0	0	0	0	0	0	2	2	0	0
Sundaram	4	1	1	1	1	1	0	0	0	0	0	2	2	3	1	0
banu	3	2	1	0	8	0	0	0	0	0	0	0	0	1	0	0
Nagarajan	5	1	1	1	1	1	0	0	0	1	1	3	2	3	0	1
Mohan	4	1	1	0	1	1	0	0	0	0	1	2	2	2	0	0
Dhavamani	6	1	0	1	2	0	0	0	0	0	0	0	1	2	0	0
Vijayan	4	1	1	1	2	0	1	0	0	0	0	0	2	2	1	0
Shahul	3	1	1	1	1	1	0	0	0	0	0	1	2	2	0	0
Arunkumar	2	1	1	0	2	1	0	0	0	0	0	0	1	1	0	0
Chandra	3	2	0	1	2	0	1	0	0	0	0	0	1	2	0	0
Kandhasamy	4	1	1	0	6	0	0	0	1	0	0	0	1	2	0	0
kalimuthu	3	1	1	0	1	1	0	0	0	0	1	1	2	2	0	0
Kannamma	5	2	1	0	2	0	0	0	0	0	0	0	0	1	0	0
Gurusamy	5	1	0	1	2	0	1	0	0	3	0	0	2	3	1	0
Muthammal	4	2	1	0	3	0	1	1	0	2	0	0	1	2	0	0
Krishnan	3	1	1	1	1	1	0	0	0	0	0	1	1	2	0	0
Veeraiyan	6	1	0	1	1	1	0	0	0	2	1	3	3	3	0	1
Palaniyamal	5	2	1	1	2	0	1	0	0	0	0	0	1	2	0	0
Chitra	3	2	1	0	8	0	0	0	0	0	0	0	1	2	0	0
Palani	4	1	1	0	1	1	0	0	0	0	1	2	2	2	0	0
Devi	2	2	1	0	8	0	0	0	0	0	0	0	0	1	0	0
Thangarasu	5	1	1	0	2	0	0	0	0	0	0	0	1	2	0	0
Senthil	4	1	1	0	1	1	0	0	0	0	1	2	1	2	0	0
Manikkam	5	1	1	1	1	1	0	0	0	0	1	2	2	3	1	0
Ajith	2	1	1	0	3	1	0	0	0	0	0	0	1	1	0	0
Pandiselvi	4	2	1	0	2	0	0	0	0	0	0	0	1	2	0	0
Duraisamy	4	1	0	1	1	0	0	0	0	0	0	1	1	2	0	0
Veerasamy	5	1	1	1	1	1	0	0	0	0	0	1	2	2	0	0

Key to Master chart :

Age :

1- less than 20 years

2 - 21 to 30 years old

3 - 31 to 40 years old

4 - 41 to 50 years old

5 - 51 to 60 years old

6 - 61 to 70 years old

7 - greater than 70 years of age

Sex: 1 – Male ; 2 – Female

Hematemesis : 0 – Absent ; 1 – Present

Melena : 0 – Absent ; 1 – Present

Risk factors : 0 – Absent ; 1 – Present

Comorbidity : 1 – CKD, 2-IHD, 3- HF

Child pugh score : 0 – Not applicable; 1- class A, 2- class B 3-class C

Rockall score: 0 – score ‘0’ ; 1 - score ‘1to3’; 2-score ‘4-7’;
3-score >7

Glasgow Blatchford score: 1- score ‘0’ ; 2 – score ‘<5’ ; 3 – score ‘>5’

Rebleed: 0 – Absent; 1-Present

Mortality: 0 – Absent; 1-Present